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TWO PEDIGREES OF HEREDITARY OPTIC ATROPHY

BY

C. H. USHER

ABERDEEN

Pedigree A. is a pedigree of Leber's disease containing three affected males in one sibship, an affected first cousin, and certainly two—one a female—but probably four affected cases in previous generations. The three brothers with the condition are members of a sibship of ten in which four are males and six females. In order of birth they come first, second, and fifth. The vision of the first-born failed at the age of 28 years, of the second-born at 21 years, and of the fifth-born at 40 years. The first-born is unmarried while the other two married and each had two children. The vision of their cousin IV 18, who is married but without issue, failed at a much earlier age than that of any of the others in the pedigree, namely, when he was 7 years of age. He is the first-born in a sibship of three, of the remaining three cases III 17, first-born in a sibship of two, married twice and by his first wife had a family of three, and by his second wife a single child. His vision failed at the age of 30 years. III 9, third-born in a sibship of eight, married and had no issue. His vision failed at the age of 30 years. III 8, second-born in the same sibship was unmarried. Her vision failed at the age of 45 years, accompanied by ptosis of the right upper lid. Catamenia continued for some years afterwards. II 5, second-born in a sibship of five, married and had two children. His vision failed at the age of 30 years. The ages of the affected individuals at the time when their vision failed range from 7 to 45 years. Of the four cases in generations II and III vision began

to fail in one of them at the age of 45 years and in the others at 30 years of age. In the three affected brothers of generation IV failure of vision occurred, taken in order of birth, at the ages of 28 years, 21 years, and 40 years, and in their cousin at 7 years of age. The order of birth in the sibships in which the eight cases of optic atrophy occur is as follows: three were the first-born, three came second, one third, and one fifth in order of birth. Six of the eight cases married and three of these had no issue. The four cases of optic atrophy with families had 10 children, of whom 6 were males and 4 females, and none of them had the family visual defect. There was no consanguinity in the parents of the affected individuals. The five sibships in this pedigree containing cases of Leber's disease had in all 28 members—11 males and 17 females—divided as follows in the individual sibships: (1) 10—4 ♂ and 6 ♀, (2) 8—1 ♂ and 7 ♀, (3) 5—2 ♂ and 3 ♀, (4) 3—2 ♂ and 1 ♀, (5) 2—2 ♂ and 0 ♀. Visual illusions were experienced by two cases (IV 18 and IV 9) and a positive central scotoma was sometimes observed by another case (III 17). The age at death of II 5 was 67 years and of III 9 47 years.

In IV 18 nystagmus was observed but only during ophthalmoscopic examination. Retinal haemorrhages were present in both eyes close to the optic disc in a case (IV 9) seen three months after the onset of the visual defect in the right eye and one month after the onset in the left eye. In a second case (IV 8) there were appearances resembling haemorrhages in the retina two months after the onset of the visual failure. In the cases of Leber's disease that were examined acuity of vision ranged from ability to see hand movements to 6/24. In comparing the conditions found after an interval of nearly eleven years in the case of IV 9 with those that were present at the first examination, vision was found to have deteriorated in the left eye, but this had occurred within eighteen months of the first examination. The periphery of the fields of vision, full at first, showed later a minor degree of contraction. The optic discs had become very pale all over, whereas at first the left optic disc seemed too red and both optic discs had shown signs of optic neuritis. The marked pallor had occurred within eighteen months of the first examination. In the case of IV 12 an examination of the blood including a Wassermann reaction and a skiagram of the sella Turcica were made.

Notes of the Cases

IV 9, *R.G.*, male, aged 21 years, seen on January 28, 1899, clerk, the first member to become affected in sibship, noticed vision of right eye failing three months ago and of left eye one month ago. Has had no illness, no headache, feels perfectly well, no history of syphilis, throughout winter had bathed in the sea

in the early morning. Grasp equal, knee jerks present, free breathing through each nostril, no nasal discharge. Eyes: pupils, equal, contract to light, eye movements full, nothing abnormal felt in orbits; R.V.=counts fingers at one foot; L.V.=5/60 not improved by spherical lens, reads 10 J. at nine inches, field of vision full. Ophthalmoscopic examination: R.O.D. too pale, white lines along edges of vessels, cup filled in, edge of O.D. blurred. L.O.D. too red, edge blurred, white lines along vessels; after homatropine next day; R. a few small haemorrhages seen at inner edge of O.D. and black pepper appearance at Y.S., L. fine haemorrhages just beyond edge of O.D., Y.S. has black pepper appearance, on nasal side of fundus at 4 O.D. diameters from O.D. are two black square patches of pigmentation whether in retina or choroid is uncertain and excepting an occasional yellowish grey spot a little larger than those in Tay's choroiditis, fundi are otherwise normal. Urine: sp. gr. 1030, no albumen, no sugar. June 18, 1900: R.V.=counts fingers at four feet; L.V.=1/60. During the test each eye turned nasalwards; optic discs pale. Nov. 16, 1900, has subjective visual sensations chiefly at centre of fields, seen both in daylight with eyes open and at night with eyes shut. The appearances take various forms as "irregular patches of red paint" or "a little blue-black spot with a sparkle in it" and other forms. R.V.=counts fingers at two feet; L.V.=1/60, fixes with nasal portion of each retina, only extreme periphery of each field is used; oph.: R. and L. media clear, pallor, almost paper whiteness, of whole of O.D., cup partially filled in, retinal vessels of good size, edge of O.D. fairly well defined; smell normal. To give up tobacco. Dec. 18, 1909, subjective sensations continue in centre of field, cannot describe shape, but they are dark or red. R.V.=counts fingers at two feet, L.V.=1/60, oph.: appearance of O.D. in each is the same in every respect as on June 18, 1900. Absolute central scotoma for 20 mm. white square in each field. In right field it extends 20° upwards and downwards, 18° nasalwards, 20° temporalwards, and 35° up and out. In left field it extends 18° upwards and downwards, 20° temporalwards, 30° nasalwards; periphery of each field is not quite full, takes little alcohol, only beer. His son V 6, and daughter V 7, are school children (Aug., 1926), with good vision and normal eyes.

IV 8, *A.G.*, male, aged 28 years, seen on May 26, 1904, married without children, was the second member in the sibship to lose his sight. Difficulty in reading for two months and in seeing at a distance for three weeks; sees best in dim light, had a full feeling in his brow, smokes 2½ ounces of a mixture one-half of which is twist, frequently drinks five or six glasses of whisky in an evening, sleeps well, appetite good. On examination: pupils, equal, contract to light, eye movements full; R.V.=6/60; L.V.=4/60;

absolute central scotoma for a 10 mm. red square in each, in right field it extends for 10° in all directions except on nasal side where it reaches 7° or 8°, in left field it extends in all directions for 10°, except on temporal side where it reaches 19°, periphery of fields full; oph.: R. and L. O.D. too red all over, but especially at temporal part; above and below each O.D. in the nerve fibre layer and near the large retinal blood-vessels is an appearance resembling much dilated small blood-vessels, or minute haemorrhages, edges of optic discs blurred at all parts, yellow spots normal; grasp good and equal; knee jerks readily obtained; tremor of tongue, not of fingers; smell, tested with iodoform, asafoetida, etc., is normal. No nasal discharge, no tender spots on head or pain on pressure; is an exceptionally strong man. June 20, 1904, tobacco has been discontinued, yet vision is worse, R.V.=5/60; L.V.=4/60, oph.: not much change, but R.O.D. is now paler at outer part than on rest of disc. Nothing abnormal found in the rest of his nervous system by Dr. A. W. Mackintosh. October 15, 1904: V.=2/60 in each eye; fields full with central scotomata; oph.: outer part of each O.D. too pale, edges not quite so well defined as normally. On May 26, 1908, his sister, IV10, reported that his vision was no worse.

IV 12, *F.G.*, male, aged 40 years, seen on January 29, 1924, was the third member of the sibship to become affected; vision had gradually failed in both eyes for six months, left eye was first affected, vision was worse on a bright day. He is in good health, subject to headaches all his life, no giddiness, no sickness, tobacco given up for five months by doctor's orders, was working hard and had worry when defect began. He experiences colour sensations when his eyes are closed: (1) flashes of all colours in form of carpets or curtains, (2) a diffuse reddish yellow colour seen when in a dark room, this lasts for a few minutes. On examination: pupils, equal, contract to light, tension normal, R.V.=1/60; L.V.=2/60, absolute central scotoma for red 20 mm. square; in right field red was recognized only in lower part, the scotoma extended from 30° below fixation point and a 20 mm. red square was not recognized again above this point; the scotoma in left eye was similar, but scarcely so large, both fields showed some peripheral contraction of temporal halves when a 20 mm. white object was used, and the right field in addition had a large wedge-shaped gap up and in from the horizontal line upwards for 60°, its apex was 15° from fixation point. The left field was full except at outer part where it was contracted to 75° at the horizontal line; a McHardy perimeter was used; oph.: R.O.D. too pale at outer part, L.O.D. has a much better colour than the right one, though temporal third is too pale. His family doctor reports that an examination of the blood had been made and it was found to be normal, and Wassermann reaction negative;

radiologist reports that "the sella is 12 mm. in its greatest antero-posterior measurement and 8 mm. deep. Its outline is regular and the clinoid processes show nothing abnormal." Feb. 20: R.V. = 1/60; L.V. = 1/60, scotomata have same characters as on last examination, periphery of fields more contracted, they both show contraction at nasal part, more in right than left field; eye movements full; smell, tested with cloves, iodoform and peppermint, is normal; optic discs of same appearance as on last examination; fifth cranial nerves, motor and sensory, normal; seventh nerves normal; hearing normal; tongue protruded straight; soft palate moves normally; gait and movements of upper extremities are normal.

Other members of the sibship: IV 10, B.G., female, aged 29 years, seen on May 26, 1908, complains of a floating black spot, no pain in eyes and no headache, occasional indigestion, bowels and catamenia regular, looks healthy, eye movements full, R.V. =6/6 Hm. 0.75D.; L.V.=6/6 not fully, with +1D. sph.=6/6; oph.: with dilated pupils after mydriatic, right eye media clear, fundus normal, left eye media clear, fundus shows pigmentation at upper-inner and inner parts most marked near periphery, but extending nearly to optic disc. The pigment is not very dark and some of it lies in front of retinal vessels; retinal vessels are normal; choroidal vessels rather more conspicuous in the pigmented area than elsewhere, fields of vision full, no scotoma detected in any part of the fields; no night-blindness. Three months ago had severe pain in both temples lasting for two hours, then nose bled and pain was relieved, had a second attack after two weeks with a swimming feeling in head, and hands felt powerless, like a faint, became very pale afterwards, recovered. Her son, V 8, has good vision. IV 11, H.G., died of typhoid, aged 21 years; IV 13, J.G., aged 24 years (1908), has good vision, and each of her three children; V 11-13, see 6/6 with each eye (1926) and have normal fundi; IV 14, and her family, V 14, 15, and 18, are abroad; V 16 and 17, twins, died in infancy; IV 15, and her son, V 19, have normal fundi and vision =6/6 in each eye of both; Dr. A. J. Ballantyne (Glasgow) kindly examined the next four cases. IV 16, aged 36 years, had no defect in central colour vision, normal fundi, vision=5/5 anl 1 J. in each eye, also V 20 and V 21 had normal fundi and no defect in central colour vision, vision =5/5 and 1 J., except in left eye of V 21, which was 5/6; V 22, aged 19 months, fundi normal. Ballantyne adds that they are all thoroughly healthy and present no anatomical defects. Father, III 6, had good sight, died, aged 59 years, no blood relationship with his wife, III 10. II 1 and II 2 had good sight, they were consanguineous, but in a degree more remote than that of first cousins. I 1, III 5 said, died at 104, could read to the last. III 1 and III 3, died unmarried, had good sight. III 2 and his family

IV 1 are abroad, none of them has defective vision. III 4, died, had good sight. IV 2, recently married, sees well. IV 3, killed at age of 24 years, had good vision. IV 4 and her daughter, V 1, aged 20 years, see well. IV 5 and her family V 2-5, the oldest is aged 14 years, all see well. III 5 has excellent vision and his ancestors for generations had good sight. IV 7 sees well. IV 6 died, aged 15 years. Mother, III 10, and five of her sisters, namely III 7, 11, 12, 13, and 14 have good sight, III 13, however has one defective eye. III 13, Mary M., R.V.=6/6; oph.: normal; L.V.=20 J. at nine inches; refraction R.H., L. my., oph.: L. choroidal atrophy adjoining O.D. and a few circular white spots in posterior part of fundus. She attributes the defect in left eye to a blow with a snowball in childhood.

III 8 a sixth sister of III 10, unmarried, intelligent, well educated female, aged 82 years (1927), with retentive memory, seen, lost her vision in the space of a few months at the age of 45 years, at this time she suffered much from headaches; catamenia continued to appear for some years after vision failed; she is second-born in the sibship of eight; drooping of right upper lid occurred at time of visual failure. On examination: a healthy active woman with 2/3 ptosis of right upper lid; pupils, equal, contract to light; tension normal; R.V.=hand movements at three feet; some contraction of field of vision (tested by hand movements), absolute central scotoma for white object of large size; L.V.=hand movements at three feet; field of vision full, absolute central scotoma for large white object—a red object, size 7 inches by 4½ inches seen at periphery of fields as brown; no colour recognized in either field, but she knows when a colour is light or dark. Her colour vision was formerly very good; oph.: R. and L. media clear, O.D. pale, but not markedly so, some narrowing of retinal vessels, edge of O.D. not sharply defined, no lamina cribrosa seen, surface of O.D. dull in appearance; Y.S. and rest of fundus normal.

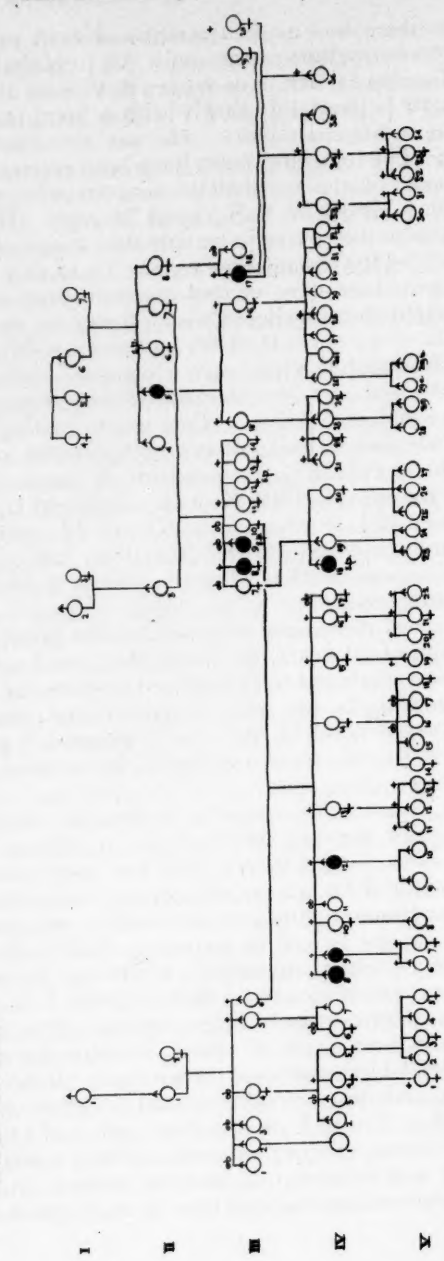
III 9, married, had no issue, died aged 47 years; at age of 30 years sight failed rapidly in both eyes at the same time and became very defective; he was a big strong man, a sail-maker, and though he continued to attend his business he was very helpless to the end of his days. II 3 drank heavily, sight good. I 2 and I 3 had good sight. The latter III 8 knew personally.

IV 18, A.S., male, aged 44 years, married, no issue, says in a letter that his vision began to fail about the age of 7 years. When first examined at age of 33 years he was told that his hereditary trouble would be transmitted by females to males, females being practically immune, "that is to say, my sisters' children run greater risks than those of my brother, especially as they are all boys." A copy of a certificate given to him by an ophthalmic surgeon on January 12, 1918, who had seen him also on May 28,

1917, shows that there was marked atrophy of both optic discs, slight persistent nystagmus seen only on ophthalmoscopic examination, refraction H. 2D. in each eye; R.V. with glass 14 J.; L.V. with glass 16 J., read 4 J. slowly with a hand magnifying glass in addition to his spectacles. He was also examined on August 28, 1915. The following notes have been received, copied from the case-book of the ophthalmic surgeon who examined him at that date: "Mr. A.W.M.S., aged 34 years. Hereditary optic atrophy. Vision discovered to be imperfect at age of 7 years, and away from school for two or three years. About this time first ordered glasses and these were revised fourteen years later. At this time he was told that he should wear glasses for near vision +2.50 sph. and later +2 sph. R. 6/60, with +2=6/24; L. 6/60, with +1.50=6/60 doubtful. Oph. very white discs with clear-cut edges, vessels of good size, no choroido-retinal changes seen, pupils smallish, equal and active. Can nearly distinguish red but it looks much paler (rose colour) opposite the centre of each eye. Peripheral field (fingers) quite full all round. Patellar reflexes normal. January 15, 1916, R. with +2.50 and L. with +2 reads 8 J., 10 J., and 12 J. slowly, with +20 reads 2 J. fairly well." IV 19 and his family V 23-24, also IV 20 and her family V 25-27 have good vision, age of V 23 is 14 years, and of V 25 is about 12 years; he wears glasses.

II 5, *W.B.*, farmer, died, aged 67 years, vision failed in both eyes at once at age of 30 years, he could never read again, his defective vision was attributed to lifting heavy stones, he did not smoke much according to his son's statement and not at all according to his nephew III 17, his eyes "quivered," probably nystagmus. The defect of vision was said to be the same as that of III 17 only worse.

III 17, aged 65 years (1926), retired schoolmaster, seen, vision failed rapidly at age of 30 years, left eye was first affected and the right eye one or two months later. He was told to leave off smoking and this he did for a year without any improvement of vision; he had never smoked much, he is a healthy and intelligent man, he says he can see as well as anyone at dusk; with a gun he can kill birds on the wing and animals when they are moving, but not when they are stationary. R.V.=2/60; L.V.=3/60; refraction H. of low degree in each; large absolute central scotoma for red and green in both fields of vision, for blue the scotoma is smaller, a 10 mm. object was used; when he is "livery" there is a positive dark and quite circular central scotoma, fields of vision are full; oph.: R. and L. media clear, pallor of O.D., but not very marked, lamina cribrosa exposed, retinal vessels of full size, edge of O.D. well defined, macula lutea normal. Although he had such defective vision he was able to carry on his work

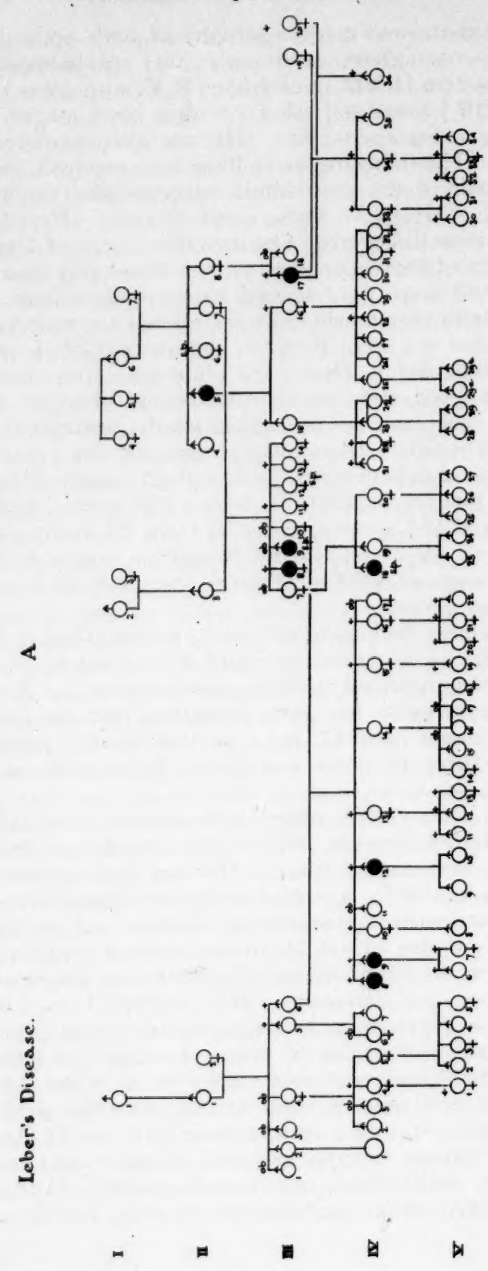


Leber's Disease.

as schoolmaster until the age for retiring. He married twice, by his first wife, III 19, he had a family of three, IV 33-35, and by his second wife, III 20, a son, IV 36, whose vision is 6/6 in each eye. All his children and grandchildren have good vision. V 30, aged 8 years, and V 31 have normal fundi. V 32-34, a family of young children with normal fundi.

III 18, farmer, with good vision, died a few months ago (1926). II 8 was the only legitimate member of the sibship II 4-8, though the other members had the same parents, II 4 died at age of 30 years. II 6, married, was without issue. None of the descendants of I 4 and I 5 is known to be affected, but not much is known about them. I 6, died aged 80 years, could read newspapers. I 7 believed to have had good sight. III 15, farmer, with good vision, fundi normal. IV 21, farm-worker, hair fair and formerly very fair, irides grey, no nystagmus, pupils sometimes red, fundi normal and pale, R.V.=6/9, refraction H. 2D. in one meridian and 5D. in opposite meridian; L.V.=6/9 not fully, refraction H. 1D. in one meridian and 4.5D. in opposite meridian. IV 22 fundi normal, vision good. IV 24, unmarried, abroad. IV 23, twin with IV 24, and his two sons V 28 and V 29 see well. In 1904, III 16, with good vision, brought her son, IV 28, aged 14 years, for examination because she thought he perhaps had the family eye affection. For two months he had been having difficulty in reading at school, though his vision was now improving. On examination R.V.=6/6 no Hm., 1 J. at 17 inches; L.V.=6/6 no Hm., 1 J. at 22 inches, fundi normal, his health had been good, no sore throat, urine, clear, no albumen, knee jerks not elicited, diagnosis cycloplegia. IV 25-32 see well. A letter from their mother, III 16 (Sept. 9, 1926), indicates that vision has not failed in any of her children. IV 25 has two sons, V 29a and V 29b, the elder is two years of age, both see well. IV 26-32 are all unmarried. IV 26 and IV 32 use glasses.

Pedigree B, contains sixteen individuals with double optic atrophy, of these fourteen are males and two females. An ophthalmoscopic examination was made in thirteen of the cases, and in the three remaining cases the histories warrant the assumption that the condition present was one of the same nature as that found in the others. All the affected individuals occur in two generations, namely, generations III and IV. The ten cases in generation III are the offspring of five unaffected sisters who presumably carry the condition. The oldest of these sisters, II 7, has four daughters and four sons, of these all of the sons are affected and none of the daughters. The next sister, II 8, had a family of nine of whom four grew up, two sons and two daughters, one of her sons was alone affected. The next sister, II 10, had fourteen children and nine of them grew up, two males and seven


Leber's Disease.
A
I
II
III
IV
V
No Consanguinity.
sp = Married without Offspring.
+ = Seen.
ob = Deceased.
IV. 8 should have s.p. below it

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females, of these one son and two daughters were affected. The next sister, II 13, had a son and six daughters, of these the son alone was affected. The youngest sister, II 18, had two sons and two daughters, of these one son died in infancy and the other was affected, neither of her daughters had any visual defect. The six cases in generation IV are all sons of unaffected females who occur in two sibships containing affected males, so that the sons have one or more affected maternal uncles.

Some of the cases marked as hereditary optic atrophy in the pedigree chart require consideration as to grouping, and more particularly those in which there is absence of a central scotoma, failure of vision in very early life, or nystagmus; and also some cases that were erroneously diagnosed as (1) tobacco amblyopia, (2) double optic atrophy cause undetermined, and (3) pressure of tumour on the chiasma.

No central scotoma was demonstrated in two instances, cases IV 10 and IV 25. The absence of such a conspicuous symptom may well cause some hesitation in grouping such cases with Leber's disease, but it has been usual to accept such cases. In one of Leber's own cases[1], a male, aged 21 years, with vision reduced to counting fingers with each eye, a central scotoma could not be demonstrated in the left field of vision on first examinations, but it was found later. Knapp[2] found a case of hereditary optic atrophy in a grandfather with symmetrical central scotoma and full fields, an affected daughter and her three affected sons had no central scotoma and the peripheries of the fields were concentrically contracted. Hirsch[3] records the case of a boy with hereditary optic atrophy in which no central scotoma was demonstrable; other three cases examined, a first cousin and two uncles, had central scotoma. Worton[4] found no central scotoma in two cases of hereditary optic neuritis. There were eleven cases in three generations and he examined four of them. Clemesha[5] in a pedigree of Leber's disease reported a mother and son with no central scotoma. Alexander[6] found no central scotoma in three brothers with hereditary optic nerve disease. Groenouw[7] found that in two-thirds of the cases there was a central scotoma only for colours or also for white. These observations are sufficient to show that absence of a central scotoma is not uncommon in cases of optic atrophy occurring in pedigrees of Leber's disease, and the two cases under discussion are, therefore, included in the pedigree as examples of the same form of optic atrophy as obtains in the cases with central scotoma.

Failure of vision in very early life. The history of the defect of vision in case IV 10 suggests that the onset occurred in early childhood, or that the visual defect was present even at birth. As

the earliest age at which the onset of visual defect occurred in the other fifteen cases was 18 years the question arises whether case IV 10 should be regarded as the same disease as the other cases, or should be discarded on account of early age at onset. Conflicting opinions have been expressed regarding cases with onset in early life. Nettleship[8] in the Bowman lecture said that "cases of family or hereditary congenital optic atrophy have been described as if forming a group in some way distinct from Leber's disease. I believe that most of these are true Leber's disease setting in very early in life or perhaps sometimes before birth." More recently Drexel[9] has expressed a different view. He regards early onset as being strongly against Leber's atrophy. For the present this case, IV 10, will be retained in the same group as the other cases of optic atrophy in the pedigree.

Nystagmus, in case IV 25, was observed during ophthalmoscopic examination in the left eye of a coal miner. The movements were vertical and not rapid. The right eye showed no nystagmus. He indicated that none of the men in the pit in which he worked had miners' nystagmus. One case, IV 18, in pedigree A. also had nystagmus. The condition is not unknown in the literature of hereditary optic atrophy. Doyne's cases with nystagmus have been published by Nettleship[8]. Waardenburg[10] saw two children in the same family with familial optic atrophy and horizontal nystagmus. Pines and Tron[11] examined four of five brothers with Leber's disease and three of these had horizontal nystagmus of both eyes besides asymmetry of face and other abnormalities. Behr's cases of complicated hereditary familial optic atrophy of childhood also had nystagmus.

Tobacco amblyopia has not uncommonly been diagnosed erroneously in cases of Leber's disease and it is probable that this happened in case III 31. In this case, a man, aged 35 years, at onset of the visual defect in which double optic atrophy was present, sight did not improve when tobacco was discontinued. The ophthalmic surgeon who saw the case regarded the defect of vision as due to tobacco, but he was unaware of visual defects in the relatives and the presence of hereditary optic atrophy in nine of the patient's first cousins. Presumably central scotomata were found but no particulars of the fields of vision could be given.

Double optic atrophy, cause undetermined was the diagnosis made by an ophthalmic surgeon in the case of IV 83, deceased. The patient had been told elsewhere that his visual defect was a family affection. His age, 25 years, at the onset of blindness, his sex, and the fact that vision did not improve when smoking was discontinued, all support the view that the case was one of Leber's disease.

Pressure of a tumour on the chiasma in case IV 92 was on first

examination, twenty-four years ago, thought to be possibly the cause of the optic atrophy. The man is now alive and well. Bruner's[12] case of hereditary optic atrophy was regarded by Dr. Hoover as probably a case of brain tumour. That pressure of a tumour on the chiasma should have been diagnosed in some cases of Leber's disease is not surprising for bilateral central scotoma occurs as an early sign in tumour at the chiasma, as has been recorded by Nettleship[13]. A case with late onset of absolute symmetrical central scotoma in a male, aged 54 years, came under my own notice in which the autopsy revealed a very large, centrally situated, brain tumour, an endothelioma, that involved both frontal lobes and pressed on the chiasma. The optic discs were atrophic and had at no time shown evidence of neuritis.

This pedigree, in which some of the cases were not at first recognized, illustrates clearly the practical use for clinical purposes of information obtainable from a pedigree that has been fully traced out and from which a clue may sometimes be found to the correct diagnosis of a case. There is little doubt that had the family history and chart of this pedigree been available when certain of the cases were examined these would have presented less difficulty in diagnosis. Still, apart from any knowledge of the pedigree, a careful examination of the fields of vision by modern methods along with other clinical evidence should usually prevent confusion in distinguishing Leber's disease from tobacco amblyopia or from cases of central scotoma arising from pressure on the chiasma.

Marriages. Eleven of the sixteen cases of optic atrophy married. Two of these were intermarried. They, as well as a third case, had no issue. This leaves eight individuals who had offspring. The largest family contained ten members and the combined families numbered 37 individuals, giving an average for each family of 4.62. The intermarriage of two individuals affected with Leber's disease is a very rare occurrence. Drexel in 1922 could find in the literature no such occurrence. In this pedigree III 6 married his first cousin III 20 and both were affected by Leber's disease. There was no issue.

Age at onset. In generation III the ages of those affected at the time of onset of the optic atrophy were 33, 31, 19, 25, 20, 30, 22, 18, 20, and 25 years, giving an average of 24.3 years. In generation IV the ages at the time of onset were 38 and 25 years, early childhood or at birth, 21, 28, and 22 years, giving an average of 22.3 years when case IV 10 is included and his vision is regarded as having been defective since birth. When, however, case IV 10 is omitted for the reason that the time of onset of his defect of sight is not exactly known and only five cases in this generation are considered, then the average age is 26.8 years.

There are two sibships containing more than one case of Leber's disease. The first one, III 19-30, has three affected members and their ages at onset of visual defect given in order of birth are 33, 31, 19 years, thus showing anticipation. In the second sibship, III 1-8, are four males affected, and their ages at the onset of visual defect in order of birth are 20, 18, 25 and 22 years, so that at onset of blindness the first-born was older than the second-born, the third than the fourth, and both the third and fourth were older than the first and second.

Order of birth of the sixteen cases. Six were first, three were fourth, and two fifth in order of birth, and there was one case in each of the following places in order of birth :—second, third, sixth, eighth, and ninth.

Length of life of affected individuals. Of five who have died four lived to a good age, namely, from 62 years to 74 years. The fifth died at 33 years after an operation for appendicitis. One case, III 5, has been lost sight of. Of the ten who are alive seven are over 50 years and four are 60 years or older. The youngest case is 40 years.

There are two *females* with optic atrophy in the same sibship of generation III. Catamenia ceased in each case at the time vision failed, at 33 years and 31 years of age respectively, and it has never reappeared. They are now 67 years and 63 years of age. Both married, the former who mated with an affected cousin had no children, the latter had one child only, a daughter with normal vision.

None of the cases showed cranial deformity. Skiagrams of the sella Turcica were made in four cases, III 20, III 22, IV 89, and IV 92, and a description of each is incorporated in the notes of these cases. Headaches were complained of at the onset of the visual defect in six cases. In reference to prognosis two cases may be mentioned that were re-examined more than twenty years after the first examination so that comparison could be made of the ocular conditions found on the two occasions. In case IV 92 at the time of his first visit vision had been failing for a year in one eye and for six months in the other, after an interval of 23 years vision had scarcely altered, as on both occasions R.V.=counted fingers; L.V.=hand movements; the optic discs, though paler, retained the same appearances as on first examination; the retinal vessels were now slightly contracted; the field of vision of the right eye had contracted peripherally to some extent and the central scotoma remained. The field of vision of the left eye remained unaltered with a functioning part limited to a small area down-in.

In case IV 89, at the time of his first visit vision had been failing for four months, after an interval of 25 years vision had improved from counting fingers with each eye to R.V.=6/12 and

L.V.=6/9; each field of vision remained full with a paracentral scotoma, and the optic discs, rather too red when seen at first, were now pale. The improvement of vision took place during six months in 1902. The first examination was made on December 13, 1901. A very marked improvement of vision such as occurred in this case has been recorded in some other cases. In Pines's and Tron's case II vision became nearly normal. Hancock's[14] case X recovered acuteness of vision=6/6 and 1J. in each eye, and his cases IX and XI recovered nearly full vision. In one of Kawakami's[16] cases the disease disappeared completely.

Absence of the affection from generation V and from the few individuals in generation VI may be ascribed to the young age of the members in these generations as the majority are not yet 17 years of age, many are females and some are descendants of unaffected males such as II 16. To economize space in the pedigree chart all the descendants of II 16, at least 78 in number, have in each generation been placed within an oblong, only the numbers of both sexes are indicated and not the sibships. No case of optic atrophy has occurred in this branch of the family.

With reference to II 3 in this pedigree who was reported to have been an albino and who had four sons with Leber's disease, and to IV 21 in pedigree A, who, though not a complete albino, showed marked signs of albinism, it is of interest to note that in a third pedigree of Leber's disease* an affected male had four albino children.

Notes of the Cases with Hereditary Optic Atrophy

IV 92, George S., aged 29 years (Dec. 9, 1903), seen, coal shoveller. In December, 1902, he noticed that vision was dim in his left eye. Objects directly in front of him could not be seen clearly with his left eye, though he could see objects at the sides. Had bad toothache on the evening before he discovered the defect. Some months later knocked against people on his left hand side owing to vision having got much worse. He noticed defect in vision of right eye at end of May, 1903, and a few weeks later was unable to read ordinary sized print. He describes the defect of vision in right eye as being similar to that in left eye at the onset of the defect, that is, there was blurring at centre of field. Some weeks after right eye began to fail he felt out of sorts:—Anorexia, feeling of weakness, occasional nausea, no vomiting, and frontal headache. No nasal discharge; "erysipelas" in right foot in August, 1903, was laid up for a week; previous to this was in bed for a few days, feeling nausea. Had been working up to time of admission to hospital in Dec. 9, 1903, when examination showed the following condition:—*Eyes*: R.V.=fingers at four feet, large central scotoma for red, with a large card only recognized the colour at lower and also outer peripheral parts of field, in other directions the object is much more clearly seen at periphery than at centre though he cannot recognize the colour; tension normal, pupils, equal, contract to light, eye movements full, corneal sensation normal. Oph: O.D. too pale, large circular cup with very pale floor, stippling well marked, edge of disc sharply cut, no white lines along retinal vessels which are not narrowed. V.S. normal also periphery of fundus. *L.V.=hand movements* at one foot, eye movements full except on attempted

* Albinism in Man, Fig. 130. Karl Pearson, E. Nettleship, C. H. Usher, see also *Trans. Ophthal. Soc., U.K., Vol. XXIX, p. CLXXXII*, 1909.

convergence when this eye does not move inwards; fundus condition similar to that of right eye except that the disc is much paler.

Previous History.—Has been strong and healthy, no syphilitic history, no constipation, does not smoke, is temperate, married, never had headaches except as stated above; never giddiness, staggering gait, or tinnitus. He is illegitimate and knows nothing of his father. His mother married someone else and had at least two children. Dr. Ashley W. Mackintosh made an examination of the nervous system. The result was almost negative. The only positive findings were some rigidity in both legs, slight Rombergism, plantar reflexes difficult to elicit, but flexor. During his stay in hospital had occipital headache once or twice. Temperature was normal throughout. Urine (Dec. 18) clear, acid, 1025, no albumen and no sugar.

March 27, 1904.—Condition practically unaltered excepting increased pallor of O.D.'s and some narrowing of retinal vessels. Fields of vision taken on several occasions on McHardy's perimeter showed on Jan. 29, 1904: R. periphery full except slight contraction below; with a 10 mm. blue square, no other colour recognized, there is an absolute central scotoma extending downwards for 12° and at sides for 10°, up-in and up-out for 20°, directly upwards no colour is seen. Left field of vision for white is practically limited to a small area in temporal half of field which extends from fixation spot outwards for 40°, down-out for 42°, downwards 26° where it encroaches for a short distance on nasal half of field, up-out it extends for 32°. On Feb. 12, right field was full, and the absolute scotoma for blue extended for 10° in lower half of field and widened upwards so that the colour was not recognized over a large area in upper part of field. In left field no colour recognized over a large area in upper part of field. In left field no colour recognized at any part. On March 27.—Right field was contracted peripherally at outer part and slightly up-in. Red, green, and yellow not recognized as such, can only say that they are not white. Absolute central scotoma for a 10 mm. blue square object extends 10° below, 40° above, 35° out, 28° in, down-out (135) 32°, down-in (120) 30°, up-out (45) 30°, up-in (45) 38°. Left field tested with a 20 mm. white square is limited to a narrow line joining the following points at 42° down, 48° down-in (120), 76° down-out (120), 32° outwards (90) 26° up-out (32). When seen again in 1926 at the age of 51 years, he said that he thought vision had got worse. Examination showed pupils, equal, contract to light, eye movements full, except left which does not move in much beyond mid-line. Marked divergence of left, no evidence of tower-skull or acromegaly. R.V. = fingers at one foot, imperfectly, in lower part of field. He worked at coal until five years ago when he had to give it up on account of the defective sight. Oph.: R. and L. O.D. very pale, greyish, edge sharply defined, retinal vessels not much narrowed, in left more than in right; Y.S. and rest of fundus normal; no white lines along vessels. Refraction low H. in each. L.V. = hand movements at six feet, limited to lower-inner part of field. Right field much contracted peripherally, 58° below, 38° above, 32° in and 80° out. An absolute central scotoma for a 10 mm. white square object extends downwards 40°, upwards 30°, inwards 28° outwards 30°. He has never smoked and does not drink alcohol. General health is good. His last and third child, a male, aged 20 years, was born a few years after his vision failed. Knee jerks equal. Report of skiagram: "The sella is regular in outline, 13 mm. A.P. and 11 mm. deep. The anterior clinoid processes are well formed, but the posterior ones are blurred and partly buried in what seems to be new bony growth. The sphenoidal sinus is well developed."

IV 89, George G., aged 22 years (Dec. 13, 1901), seen, fireman on a locomotive, noticed sight failing four months ago and that vision gradually got worse for two months and then remained stationary. Eight months ago he passed the usual railway test for eyesight. Sometime before vision failed, he had been troubled almost daily with headaches, mostly frontal, also with pain in stomach and dizziness. When smoking was discontinued two months ago the vision did not improve though it has not continued getting worse as it had been doing previously, and headache also has ceased. Does not work with lead and there are no symptoms of lead poisoning. For the last five years admits having occasionally drunk too much, but it never prevented him doing his work. Began to smoke at 16 years, and smoked about three ounces of Irish twist weekly. No history of syphilis.

Tongue and hands are tremulous; urine, amber, acid, 1020, no albumen, no sugar; no intention tremor, knee jerks present and not exaggerated, no ankle clonus, cranial nerves: I distinguishes peppermint and asafoetida, II, III, IV, VI *vid.*

infra; V, sensation of face normal; VII movements of each side of face equal; *VIII hears watch* with each ear at one foot, has not noticed hearing worse recently; IX, X, XI, XII *nothing* pointing to any affection of them. *R.V.=fingers at one* foot, full field, absolute central scotoma for red, with a 10 mm. object used, it was nearly circular *extending to 20°, refraction estimated* low H. L.V.=fingers at one foot, field full, absolute central scotoma for red, with a 10 mm. object, it extended *upwards* 10°, inwards 12°, downwards 10°, and outwards 30°, it is much larger external to fixation point than nasalwards, *refraction estimated low H, pupils*, equal, size 3.75 mm., contract to light, eye movements full, no tenderness on pressing *eyeballs backwards; oph.:* O.D.'s normal, or rather too red, deep physiological cupping, some of the smaller vessels are tortuous and those passing downwards from O.D. show abrupt antero-posterior bendings in the nerve fibre layer which give an appearance of small *haemorrhages at the parts where they* are foreshortened. Possibly there are a few minute flame-shaped haemorrhages in this region. *The Y.S. and other* parts of the fundus are normal. On Jan. 23, 1902, V.=fingers at two feet in each eye, fundus remains as on first *examination* and also pupils and eye movements. He thinks there is more mist in front of his eyes. *He fixes a point to the* left of the object he wishes to see with each eye. When seen again on Dec. 26, 1926, aged 49 years, vision had greatly *improved*. This had taken place within six months during 1902. R.V.=6/60, with +1D.=6/12, with +3.50D. reads 1 J.; *L.V.=6/60, with* +1.50D.=6/9, with 3.50D. reads 1 J. Says he cannot recognize people in the street although he can read *very small print*. Fields of vision full, large paracentral scotoma for red, green, and blue above fixation point, pupils, equal, contract to *light*, eye movements full. Oph.: R.O.D. pale, edge sharply defined, lamina cribrosa much exposed, large cup, vessels of *good size, narrowing* of inferior temporal artery on O.D., L.O.D. same appearance as R.O.D. only is paler and no contractions on vessels. *Is not* acromegalic and has no tower-skull. Has not again smoked a pipe, but is smoking 70 cigarettes weekly. Takes no alcohol, married at age of 27 years, is without issue; general health is good. Knee jerks present and equal. Report of skiagram, March 29, 1927: "The sella is not quite regular in outline and its wall is thin. The clinoids, antero and postero, are small and blurred. The sella is 14 mm. A.P. and 13 deep. There is a blurred but quite distinct shadow, lying free in the sella, as if some mass, rather dense, was lying there"

III 6, Alexander Da., sixth-born in a sibship of eight, aged 52 years (Oct. 27, 1909), seen, pensioner, vision failed at age of 25 years and it never got better, married his first cousin, A.R., III 20, who has double optic atrophy, had no issue; died in 1921 after operation for gastric ulcer. Smoked two ounces of tobacco weekly, saw best in dim light. R.V.=hand movements at six feet; L.V.=hand movements at six feet; fields of vision full when tested by hands, absolute central scotoma for white in each, colours not recognized anywhere in either field; pupils, equal, contract to light; eye movements full; oph.: R. and L. media clear, O.D. pale, right rather paler than left, retinal arteries distinctly narrowed, especially in right eye; lamina cribrosa exposed over most of the disc, the colour of disc is white rather than grey, no white lines along retinal vessels, some disturbance of pigment at edge of optic disc, especially right disc; some blood-vessels near the disc are more tortuous than usual; no abnormal exposure of the large choroidal vessels, no retinal pigmentation, nothing to suggest retinitis pigmentosa, diagnosis is Leber's disease.

III 4, William Da., aged 73 years (1926), seen, retired coal miner, is fourth in a sibship of eight, married, had a family of ten, vision became affected at 20 years of age. He is very deaf. R.V.= hand movements at three feet; L.V.=hand movements at three feet; fields full tested by hands, central scotoma in each for large white object, no colours recognized. Oph.: L.O.D. pale, retinal vessels of fair size; R.O.D. not seen; pupils were small and conditions difficult for examination because he is so very deaf and frail; smokes one ounce of bogie roll tobacco weekly.

III 5, John Da was unmarried when he went abroad and has not been heard of since, is fifth in order of birth in sibship of eight; vision failed at the age of 18 years, and though he was younger than his brother William his vision was first affected. His loss of sight is attributed to the family affection.

III 8, Robert Da., coal miner, eighth born in sibship of eight, was healthy, no illnesses, hearing good, vision failed at 22 years, and he then became a pedlar. Had a twin sister Mrs. W. (III 7). He married and had a twin son and daughter.

now aged 21 years; his wife died at the birth of the twins. He died on May 14, 1921, result of injury, aged 62 years.

IV 25, Hugh W., coal miner, first-born in a sibship of nine, aged 42 years (1926), seen, vision failed in both eyes at age of 21 years, says he is strong and has had no illness, formerly smoked heavily, but has not smoked for six years; drank heavily once a week, but for last seven years has had no alcohol; has bilious attacks that last for two or three days, these come on at intervals of from one to several months; married, has a family of four daughters (V 48-51) of which the oldest is six years. R.V.=3/18 and 6 J.; L.V.=3/36 and 20 J., fields of vision full, taken with hands; no central scotoma for colour could be detected; a 4 mm. red square object was recognized by the right eye and was seen best at fixation point; nowhere in the left field of vision was a 4 mm. red or green object seen, but an 8 mm. one was seen and the colour was more marked at fixation point than elsewhere, the examination though satisfactory was made just after he had returned from work in the pit; oph.: R. and L.O.D. pale, especially the left one, lamina cribrosa exposed, retinal vessels, of about normal size in right eye, are rather diminished in left eye; during fundus examination vertical nystagmus was observed in left eye, not in right eye. He said there was no "glennie blink" (miners' nystagmus) in the men in his pit.

IV 10, John P., coal miner, third-born in a sibship of five, aged 40 years (1926), seen, does not admit that his vision is defective, though his sister, Mrs. Wi. (IV 9), said that he was very blind, is married, has three children (V 14-16). Examined in his dwelling. R.V.=4/24, 16 J.; L.V.=4/12, 4 J., refraction emm. or low H. in each, fields of vision tested by hands show no contraction, no central colour scotoma detected in either field, pupils, equal, contract to light, oph.: R. and L. O.D. pale, retinal vessels of good size, edge of O.D. well defined, lamina cribrosa visible, diagnosis double optic atrophy no evidence of previous optic neuritis; knee jerks present. The impression he gave was that he did not wish to acknowledge any defect of vision and although it is well known to his relatives that his sight is very bad, subsequent to the examination, in reply to a letter, he sent the following: "I really do not know when my right eye became defective. As far back as I remember there has been no difference. I am sure it has been the same all my life. I have smoked black tobacco, cigarettes, and also chewed tobacco since I was fourteen years of age. I cannot remember it ever affecting my sight. I have also worked in the coal mines for 26 years using safety lamps, and I think my sight is just as good to-day as ever it was."

IV 83. William L., boxmaker, unmarried, first-born in a sibship of four, died aged 33 years, after operation for appendicitis; vision failed in both eyes at age of 25 years, had in consequence to give up his work, but did not get quite blind. He had been examined by an ophthalmic surgeon, who told me that he diagnosed double optic atrophy, but had been unable to discover the cause; no information regarding fields of vision. His aunt (III 39) said that at one time he was seen by a professor at a distance, who said it was a family disease for which there was no cure. Smoked not heavily, on advice gave it up, but vision did not improve.

III 31, John S., blacksmith, first-born in a sibship of seven, died in a poor-house in 1923, aged 74 years, causes of death certified as being "senility and cerebral haemorrhage of three days duration"; sight failed about the age of 35 years, but recovered sufficiently to allow him to get about, had to give up work as blacksmith for that of night watchman, was not a drinker, married at age of 21 years and had ten children (IV 74-82). An ophthalmic surgeon said he had taken the case for one of tobacco amblyopia and admitted that vision did not clear up when instructions were given for smoking to be discontinued and that double optic atrophy was present. Presumably central scotomata were found.

III 56, Alexander In., street porter, youngest in a sibship of four, aged 60 years (Nov., 1926), seen, vision failed rapidly when he was aged 20 years, and worsened for about nine months, he thinks that during the last twenty years vision has got very slowly worse; smoked, and still smokes one and a half to two ounces of bogie roll tobacco, always moderate with alcohol and before onset of blindness was a total abstainer; health good: when vision failed had bad headaches in temples, forehead and occiput; married twice, had two sons by first wife, a daughter and three sons by second wife: intelligent, healthy in appearance, and able to do his work which entails lifting heavy weights. R.V.=counts fingers at one foot; L.V.=counts fingers at one foot; does not fix centrally with either eye, fields of vision quite full

as tested by hand movements; large central scotoma for white in each field; red, green, blue, and yellow not recognized in any part of either field; pupils, equal, rather large, contract to light; eye movements full; tension normal; oph.: R. and L. O.D. marked pallor, lamina cribrosa exposed, edge of O.D. well or even sharply defined, retinal vessels somewhat narrowed, Y.S. and rest of fundus normal; refraction low H.; knee jerks present.

III 14, William Co., compositor, fifth in a sibship of nine, vision, good at school and even later, appears to have failed about the age of 25 years; it became very bad, though he was not blind, so that he did practically no work afterwards but loitered; health good, not subject to fits; he smoked; unmarried; died on Nov. 4, 1926, in a poor-house, aged 65 years. The medical officer kindly supplied the following information: He was admitted January 26, 1922, with chill, diarrhoea, and blindness; apart from the diarrhoea and slight temperature, with headache the only other physical signs noted were those of aortic incompetence; Wassermann negative; on February 7, 1922, discharged cured, apart from the blindness, to the main house of the poor-house; re-admitted to hospital on October 30, 1926, unconscious, doubly incontinent, and with a temperature of 102° and a right-sided hemiplegia; no post-mortem; no history of his eye condition having been thoroughly examined.

III 29, George Re., coal carrier, ninth in sibship of ten (there were four others died in infancy, order of their birth not known), aged 55 years (1926), seen, married in 1899, is without issue; vision failed at 19 years, right eye first affected, left eye soon afterwards, visual defect became stationary in about six months and has not got worse since; neuralgia in left side of face, attributed to teeth, accompanied onset of visual defect; smoked three ounces of bogie roll tobacco weekly and continues to do so, drinks beer and rum on Saturdays; had scarlet fever and enteric when in the army. Sexually no alteration when vision failed (compare cessation of menses in women); R.V.=fingers at one foot; L.V.=fingers at one foot; fields of vision full, absolute central scotoma for colours and white in each field; oph.: R. and L. O.D. pale, edge well defined, lamina cribrosa exposed, retinal blood-vessels show some localized narrowings, in left eye white lines present along some of the vessels; refraction estimated emmetropic in each eye. Sense of smell (cloves, asafoetida, peppermint) and hearing normal; knee jerks present and equal.

IV 1, William Re., labourer, unmarried, first-born in sibship of four, aged 52 years (1926), seen, vision began to fail when he was aged 38 years, got worse for about twelve months, and since then has remained stationary; pain in forehead accompanied onset of visual defect, at present can see sufficiently to walk alone in quiet back streets; told, in 1912, by an ophthalmic surgeon to leave off tobacco which he did without any improvement of vision; smoked three and a half ounces of twist and drank one or two pints of beer weekly; looks healthy, is quite intelligent, hearing and sense of smell tested and found normal, no cranial deformity; R.V.= counts fingers at one foot; L.V.=counts fingers at one foot; fields of vision full as taken by hand movements, absolute central scotoma in each field, large coloured objects not recognized in any part of fields, a 10 mm. white square was seen when moved at fixation point, but much better when removed some distance from it; oph.: R. and L. media clear, refraction estimated low H., O.D. pale, edge quite well defined, lamina cribrosa exposed, retinal arteries slightly narrowed, yellow spot and rest of fundus normal.

III 22, Mary Fo. née Re., married, fourth-born in sibship of ten (see III 29), aged 63 years (1926), seen, has an only child, a female (IV 45). Vision began to fail at the age of 31 years, at same time catamenia ceased and never reappeared; health always good; R.V.=counts fingers at one foot; L.V.=counts fingers at one foot; fields of vision full, tested by hands, absolute central scotoma for large red object in each field; refraction estimated low H. in each eye; oph.: R. and L. media clear, much pallor of O.D., edge regular and well defined, lamina cribrosa exposed, retinal arteries somewhat narrowed, no white lines along their edges; yellow spot and rest of fundus normal; report on skiagrams of sella by the radiographer: "sella is too small and there is blurring of the posterior clinoid processes by bone, sphenoidal sinus is very large. On a subsequent day another skiagram was taken when the films showed the same enlargement and deformity of the posterior clinoids as was noted in the first skiagrams. Never smoked.

III 20, Aggie Da. née Re., widow, married at age of 22 years, and had been married for forty years, no miscarriages, no stillbirths, second-born in sibship of

ten (see III 29), aged 67 years (1926), seen, vision failed about age of 33 years, had much headache referred to vertex; visual defect, noticed first when reading, in a few months got very bad; catamenia regular until vision failed, then ceased and never re-appeared; does not smoke; always had good health, no illness; sense of smell tested with peppermint, oil of cloves, asafoetida, is normal; R.V.=little more than P.L.; L.V.=hand movements at one foot; fields of vision tested by hand movements are peripherally contracted in all directions, large red object not recognized as red in any part of field, but when tested with large white object there is obviously a central scotoma in each field; oph.: R. and L., media clear, O.D. pale, edge well defined, lamina cribrosa exposed, some narrowing of retinal arteries, no white lines along their edges, nothing abnormal seen in rest of fundus; refraction estimated low H. Skiagram report: "The sella is rather irregular in outline, especially towards the posterior part. It is 15 mm. A.P. and 11 deep. Both clinoid processes are small and deformed, and are partly obscured by new bony deposits."

Unaffected Members of the Pedigree

GEN. I.—1, Da., died abroad. 2, Mrs. Du., good vision, her husband, Wm. Du., died early, not in chart, nothing known about his sight.

GEN. II.—3, John Da., soldier, "blin-fair" (albino), drank heavily, *ob.* 40. 4, killed 5, Grace Da., married twice, no issue by first and none known by second husband. 7, Mary Du. (Mrs. Da.), first-born in sibship, vision good. 8, Isabel Du. (Mrs. Co.), and husband, 9, saw well. 10, Agnes Du. (Mrs. Re.) and husband 11, sight good, he died, aged 68 years. 12, Wm. Du, unmarried, killed. 13, Barbara Du. (Mrs. Sm.) and husband, Wm. Sm., 14, had good vision, he died of cancer of tongue. 16, Peter Du., married twice, had two families and many descendants. 18, Catherine Du. (Mrs. In.), youngest in sibship, and her husband, 19, had good sight.

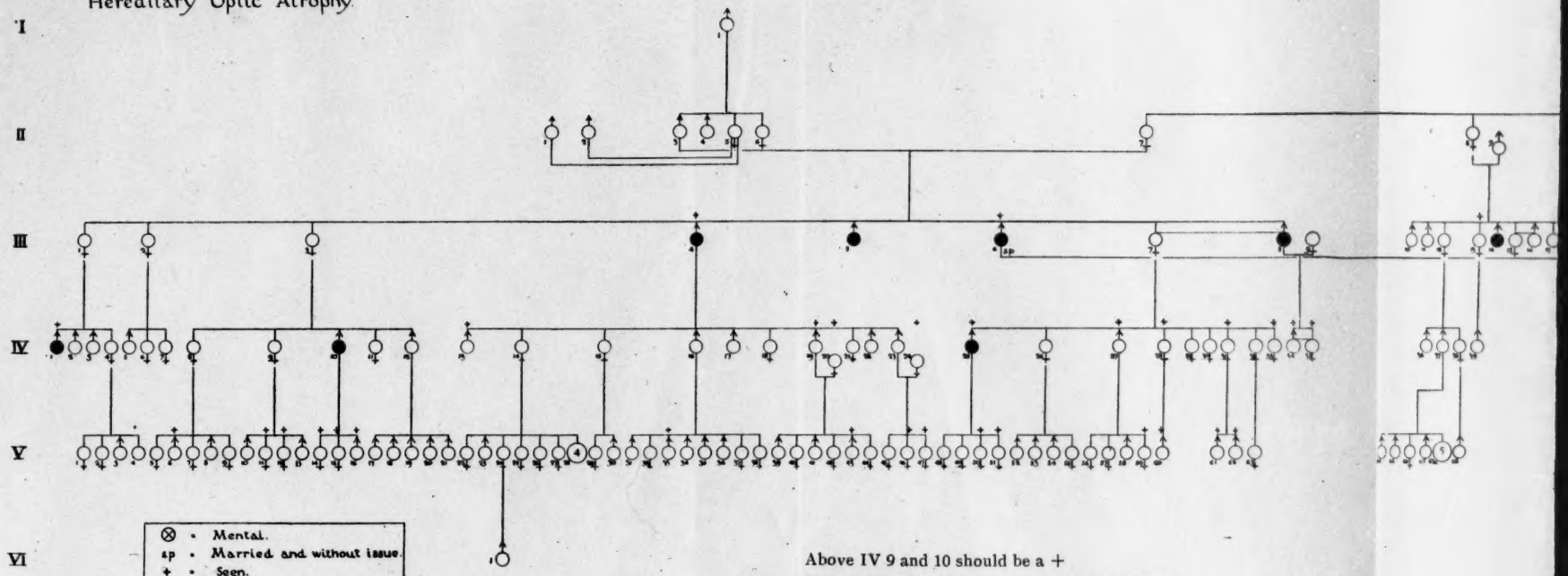
GEN. III.—1-8 family of Da.: 1, Jessie (Mrs. Re.), ob. 65 of brain trouble. 2, Mary (Mrs. MacCn.), abroad. 3, Grace (Mrs. Pa.), and 7, Jane (Mrs. Wa.), vision good, 7 twin with 8. 9, died at birth of her twins. 10-18 family of Co., 10 and 11 *ob.* childhood. 12, Mrs. Le., and 13, Mrs. El., vision good. 15, Jessie, *ob.* 16. 16, her twin brother, *ob.* childhood. 17 and 18, *ob.* infancy. Only three of this family grew up. 19-30, family of Re., 28 (4 *ob.* infancy or still-born). 23, *ob.* 12 "water in head." 26, Janet (Mrs. We.), *ob.* 27, Isabella (Mrs. An.), widow, 19 (Mrs. Al.), 24 (Mrs. Fo.), 30 (Mrs. Du.) had good vision. 21, Williamson Re., phthisical, off work 12 years, R.V. 1J. close, central nebula; L.V. $\bar{c}$+13D. 1J. readily, fundi n. 31-39, family of Sm., all saw well except 31. 32 (Mrs. Ly), *ob.* 36, had illegitimate son (IV 92) by 35, later she married 37 and had two children, IV 93 and 94. 34, age 73 years, healthy. 38, married, no issue. 39, unmarried. 40-47, first family of Peter Du. (II 16), 40, Wm. and 47, Betsy, *ob.* 42, married three times, three boys of first marriage and a girl of each of the other marriages. 45, John, and 46, twins. 46, *ob.* infancy. 45, had 14 children (IV 107-120). 48-51, second family of II 16. 48, Peter and 51, Mrs. Ra., both married no issue. 50, Wilhelmina, unmarried. 49, Charles. 52-56, family of In.: 52 Isabel, has three illegitimate children (IV 131-133). 53, Maria, has illegitimate daughter (IV 134). 54, *ob.* infancy.

GEN. IV.—1-4, Re. family. 2, John, aged 45 years. 3, Matthew, aged 43 years. 4, Grace (Mrs. Mo.), vision good in all. 5-7, MacCn. family, abroad, state of vision unknown. 8-12, Pa. family. 8, Mary (Mrs. Co.), 9, Agnes (Mrs. Wi.) and 12, James, who is abroad have good vision. 11, Jane, *ob.* aged 2 years. 13-23, Da. family all have good vision. 13, John, and 17, James, unmarried. 14 (Mrs. McEw.), 15 (Mrs. Ing.), 16, Wm., 19, Peter, and 23 Alex. have families. 25-33, Wa. family. 26, Mary (Mrs. Go.), has young family (V 52-55). 27, John, coal miner, vision 1J. in each, fundi n. 28, Minnie (Mrs. Cl.), R.V. 2J., H.As., L.V. 1J., H. 29, Grace (Mrs. Co.). 30, Jean, 31, Lizzie (Mrs. Fl.), healthy, has two boys. 32, Nettie, her child (V 63), *ob.* infancy. 33, Robina, unmarried, of healthy appearance. 34, Thomas Da., aged 21 years, seaman, vision good, 35, his twin sister, has diffuse left corneal opacity, R. vision good. 36-38, Le. family. 36, killed, aged 14 years. 37, Henry, in army, aged 40 years, says in letter, Feb. 4, 1927, his sight is quite good, has nine children, oldest is 13½ years. 38, abroad. 39, unmarried, good vision.

40-43, Al. family. 40, Annie, 41, Barbara (Mrs. Ne.), 42, Wm., married, no issue, and 43, Maggie (Mrs. Jo.), have good vision. 44, John Re., aged 25 years, scoliosis, cannot walk or read, can draw, imbecile. 45, Mrs. Hu., vision 6/6 in each, fundi n. 46-51, Fo. family. 46, imbecile, died in institution for imbeciles. 47, George, abroad, married, no issue. 48, abroad, no report of any family. 49, Elizabeth, married, 50, Lena, 51, Edwin, both unmarried, good vision in all. 52-54, We. family, 54, Joseph and 53, Mrs. Du., see well; 54, ob. 55-64, An. family, 55, Bella, 56, Nelly (Mrs. Mi.), 57, stillbirth, 58, Maggie (Mrs. St.), 59, and 61, *ob.* childhood; 60, Robert, 62 and 63, males, 64, Eva none with defective vision. 65-72, Du. family, father and mother (III 30) had same name, but not related. 65, Robert, and 66, Mrs. St., married; 67, *ob.* infancy; 69, Mrs. Macdo., married twice, only child, V 102, by first marriage, died, family of six by second marriage; 71, John; 72, Wm., aged 20 years, mentally weak. 74-82, Sm. family. 74, Wm., married twice and had two families. 76, Hugh, drowned. 77, Mrs. Du.; 78, John; 79, David, cannot smoke. 80, James; 81, Peter, abroad; 82, Bella (Mrs. No.), aged 36 years. 83-86, Ly. family. 84, Donald, baker, aged 48 years, 85, Mrs. Wi., 86, Lizzie, unmarried, all see well. 87, Harry Bu., *ob.* 26, influenza; 88, Dora Bu., married, no issue. 89-91, Gi. family. 90 *ob.*, could never learn to smoke. 91, Wm., married, no issue, vision 6/6 in each eye, fundi n. 93 and 94, Gr. family were young children 30 years ago, one died, no trace of this family can be found. 95-101, Du. family. 95, Robert, abroad, 96, Mina, and 97, Peter, have families. 99, Maggie, married twice, had two families. 101, Wm., married, no issue. 102, daughter of father's first marriage, 103, Jim, 104, Ben., and 105, of second marriage and 106, daughter of third marriage of III 42. 107-120, John Du.'s family. 107, *ob.* childhood. 108, John, had convulsions, married, no issue. 109, Wm., 110, Lizzie, 111, Mary, 114, Nelly, 115, Jim, all married with families, 112. Aggie, and 113, Alex., married, no issue. 116, *ob.* infancy, 117 and 118, stillborn, 119, *ob.*, aged 4 years, 120, *ob.* infancy. 121-124, twice twins all *ob.* young, 125, Peter, 126, Lizzie, 127, Kate, 128, Jessie have good vision. 129, male, sex of 130 unknown. 131-133, illegitimate, 131, Jean, married, no issue. 132, Kate, has family. 133, killed. 135, Wm. In., aged 35 years, married, has two sons (V 181-182). 136, Alex., unmarried. 137-140, In. family, 137, Mary In., aged 19 years, 138-140, three healthy boys, vision good.

GEN. V.—1-4, Mo. family: 1, Grace; 2, Jessie; 3, Wm., 4, another, sex unknown. 5-9 Co. family: 5, Grace; 6, James, aged 20 years; 7, Agnes; 8, John; 9, female. 10-13, Wi. family: 10 male, 11 female with epicanthus both sides, 12 female, 13 male, 14-16 are young children. 17-21, five boys, three died. 18, aged 6 years. 22-28, McEw. family: 22, 23, and 27, *ob.* phthisis. 24, Mrs. McF. has a child, VI 1; 25 Robert, 26 Janet, 28, four *ob.* infancy. 29, Elizabeth, aged 13 years; 30 Oliver, 31, Wm. lost an eye from ulceration, 32 James, 33 Robert, 34 John, 35 Charles, 36 James, 37 Lizzie, and 38 May, all see well. 39-44, 39, first-born, aged 11 years. 45, *ob.*; 46, Elizabeth; 47, May, good vision. 48-51, first-born, aged 6 years. 52-55, first-born, aged 12 years. 56-59, all young with good vision. 60 only child. 61 L. corneal opacity. 62, aged 10 years, R. int. con. strabismus, infantile paralysis right leg which is thin and foot small. 64-67a, Le. family of nine: first-born 13½, all see well, six have passed sight test at school. 68, abroad. 69, a family, no particulars. 70-72, three young girls. 73-76, four young children. 77, aged 6 years. 78, *ob.* sex unknown. 79, boy. 80-83, first-born, aged 6 years. 82, *ob.* ten weeks, living ones see well. 84-88, first-born, aged 14 years. 89-93, Mi. family; 89 Ella, 90, female, 91, male, *ob.*, 92 schoolboy, 93, *ob.*, twin with 92. 94 and 95 stillborn twins; 96 Bertie St., 97 only child. 98 and 99 boy and girl. 100 *ob.*, aged 2 years; 101 girl. 103 and 104 twins, *ob.*, 105, 106, 108, see well, 107, *ob.* 109 Fred, aged 29 years, 110 Wm., 111 John, 112 Jane, 113 Annie, 114 Frances, aged 6 years, only child of Wm. Sm.'s (IV 74) second marriage. 115, male, aged 25 years, corresponds regularly with his mother (IV 77), who says his sight is good; 116 *ob.* 117, Robert, aged 23 years, reads 1J.. 118 John, 119 Lizzie, typist. 120-126, first-born (120), aged 21 years, 124 *ob.*, fundus oculi n. in living members. 127 only child. 128-132, abroad, all see well. 133-140. 138, aged 7 years, 139, aged 4 years, 140, aged 2 years, others *ob.* infancy or in first three years of life. 141 only child; 142 *ob.* infancy.

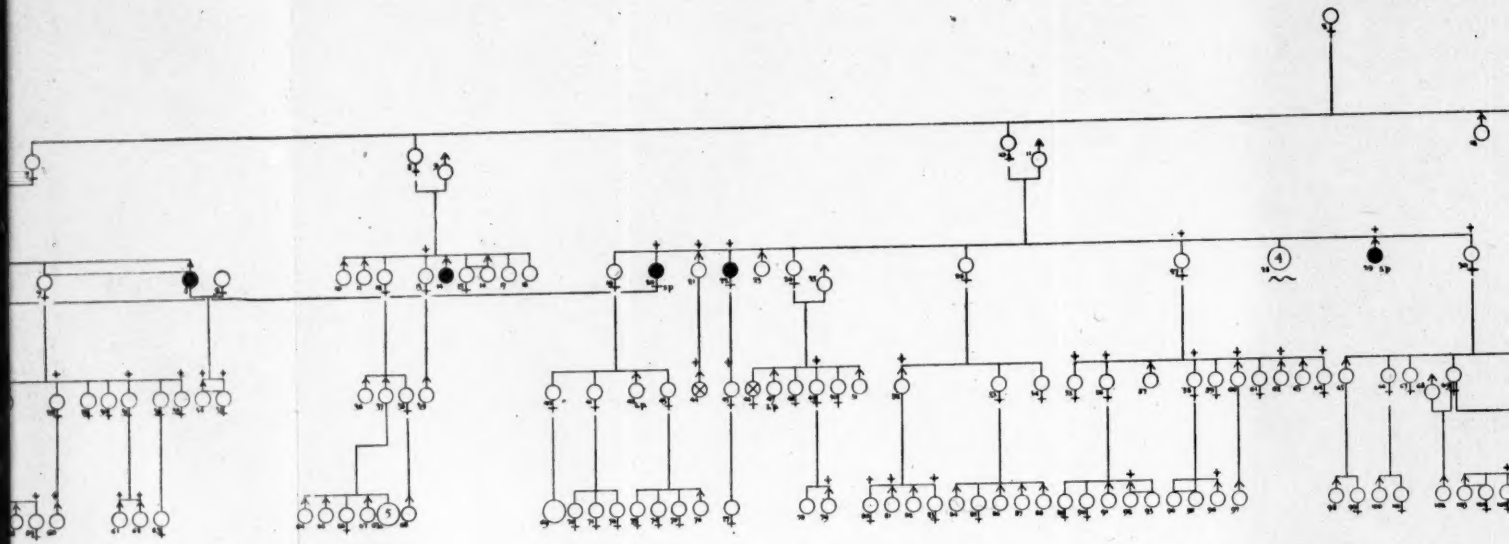
Hereditary Optic Atrophy



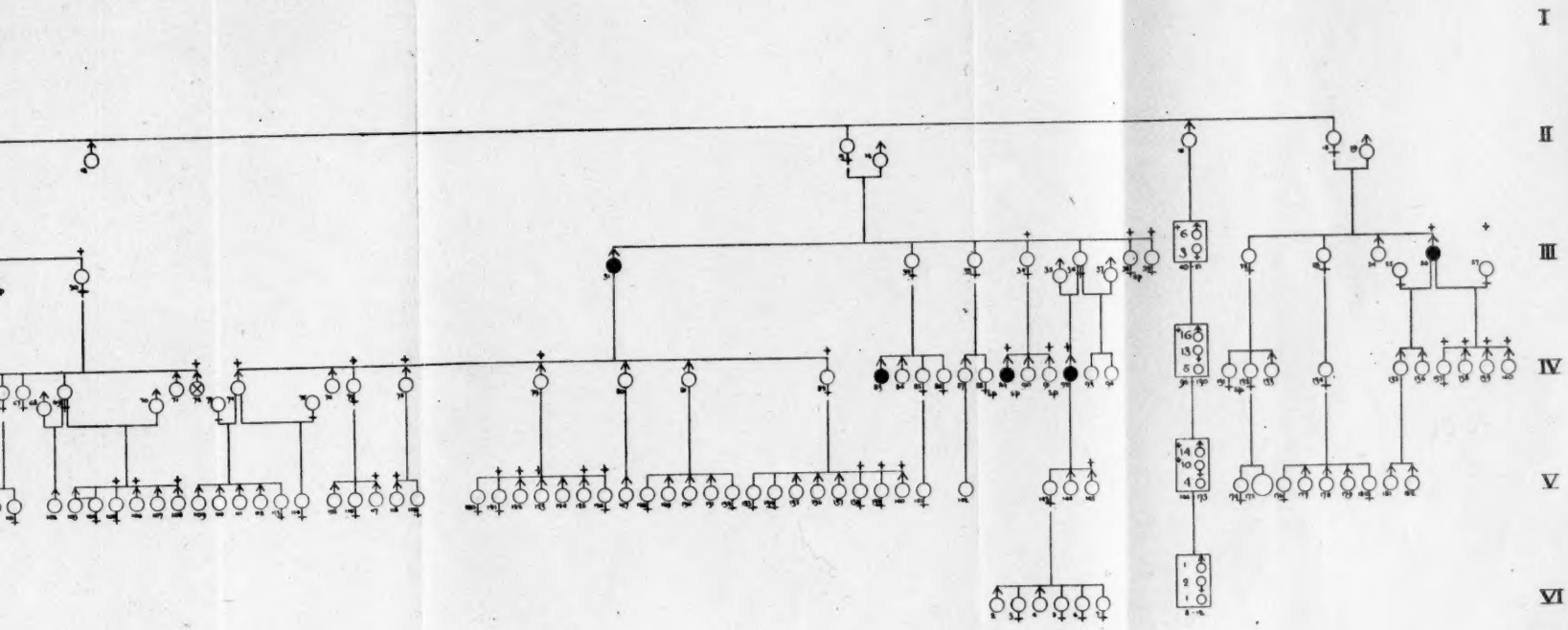
⊗ = Mental.
sp = Married and without issue.
+ = Seen.
~ = Order of birth not known
○○ = Twins.
No consanguinity of parents.

Above IV 9 and 10 should be a +
V 25 and 32 should have male *not* female sign
V 112 should have female sign *not* male

B.



sign


I
II
III
IV
V
VI

143, Barbara, aged 29 years (1916), 144 ob. infancy. 145, Charlie, aged 20 years, unmarried, fundi n., vision 6/6 in each. 146, male, *ob.*; 147 and 148 females. 149 a family no particulars. 150 and 151, sex not recorded, were first family, and 152 Marjory, 153 male, and 154 others number and sex not known, were second family of IV 99. 155-159 family of Wm. Du. (IV 109). 155 Wm. has phthisis, 156 Alex., 157 female *ob.*, 158 female, 159 male, all see well. 160-162 family abroad, 160-161 male twins, 162 female. 163-166 family abroad, 163 female, others males. 167-170, 167 Catherine, 168 Margaret, 169 Jack, 170 male. Jim Du.'s family, 171 and 172 males, 173 female, see well. 174 female, 175 others no particulars. 176-180 age of first-born is 16 years. 181, aged 5 years. 182, aged 3 years.

GEN. VI.—1, a child. 2-7, first-born aged 9 years, 2, 3, 5, 6, 7, see well, 4 died. 8, only child of Wm. Du. (V 155), died. 9 and 10 abroad, son and daughter of V 160. 11 and 12 abroad, two children of V 161, sex unknown.

These two pedigrees, A and B, were being worked up for publication before it was known that they included females. This is mentioned because it has been stated that small pedigrees with only males affected are relatively common and not always recorded, but cases in females have been more generally published on account of their rarity.

I am indebted to Dr. John R. Levack for the radiographic examinations and reports.

George G., IV 89 in pedigree B, was an in-patient at the Aberdeen Royal Infirmary in 1902 and George S., IV 92 in pedigree B, in 1904.

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A PEDIGREE OF A FAMILY SHOWING HEREDITARY GLAUCOMA

BY

R. R. JAMES

LONDON

CASES of hereditary glaucoma, though by no means unknown, are yet relatively rare, if we can judge from pedigrees that have been reported; and yet in my opinion the condition is possibly not so uncommon as would appear, and many more pedigrees could probably be constructed by careful inquiry, if we could only find time to go into the family history of each case we see.

The classical example in English ophthalmic literature is that recorded by Lawford in the *Roy. Lond. Ophthal. Hosp. Reps.*, Vol. XVII, p. 57. The latter observer saw several of my cases in consultation and it is largely owing to him that this pedigree has been collected for reproduction here.

(1) A country parson, the eldest child of a family of eight. He died a good many years ago, aged more than 60 years, and so far as is known exhibited no signs or symptoms of glaucoma.

(2) His wife was under the care of Mr. J. Herbert Fisher. She was first seen in November, 1912. She had accidentally discovered that the sight of the right eye was defective, and this had been diagnosed by the late Mr. Doyne, in the previous July, as due to glaucoma.

The vision of the affected eye was less than 6/60, the disc was pathologically cupped, and the field of vision was curtailed in an unusual way for glaucoma, the defect being especially in the lower and the temporal side. The most abrupt edge of the cup appeared to be up and in. The tension of the eye was pathologically above the normal; the pupils were equal. Eserine had been used, but did not keep the tension satisfactorily down.

On December 12, 1912, Mr. Fisher trephined the eye. It did not do very well and rather more than a week later the aqueous chamber had not re-formed. Eventually good filtration was obtained, with a shallow chamber and normal tension.

At the end of April, 1914, she had severe pain with vomiting for two days, was the subject of a quiet iritis, and the pupil was blocked. The anterior chamber was absent, and as the eye was of no value visually, painful and likely to give much trouble, it was excised in May, 1914. For the next two years Mr. Fisher was able to keep the left eye under observation, and it retained, with the correction of a small degree of hypermetropia with astigmatism, 6/9 vision. This eye exhibited no signs of glaucoma throughout. The tension kept normal and the field remained good. This lady

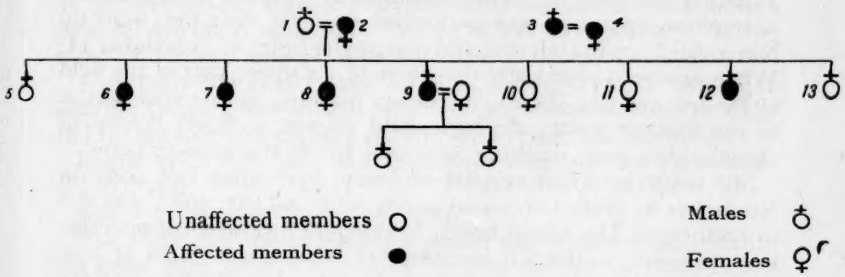
was the eldest child of a family of four, and so far as is known, none of the others showed any symptoms of glaucoma. She died in the summer of 1926, aged 85 years.

(3) This gentleman was the youngest brother of No. 1; both he and his wife (No. 4) had chronic glaucoma, but neither came under my observation, and the fact was supplied to me by one of his nieces, No. 6.

(5) The eldest son is aged more than 60 years. I am informed that he shows no signs of the disease; I have not seen him.

(6) The eldest daughter is a lady, aged 58 years. She had no symptoms of glaucoma, but came to me in September, 1926, with a younger brother, No. 11, to be examined in view of

PEDIGREE



the family history. She was a myope, and had worn glasses for forty years; she had observed no halos. She had the large eyes of a myope, with deep anterior chambers, equal and active pupils, and normal tension in the left eye, while that of the right appeared to the finger to be very slightly above the normal. Her vision was as follows:

Right eye: -5.5D. sph.—1.0D. cyl. axis, 50° in, = 6/6.

Left eye: -5.5D. sph.—0.5D. cyl. axis, 50° in, = 6/6.

With an addition of +2.0D. sph. in each, she read J.1.

The media were clear and the fundus was normal; there was a very small degree of hyperphoria present. The left field and blind spot were normal; the right field showed a defect characteristic of chronic glaucoma, up and in, and slight general enlargement of the blind spot. She was ordered to use eserine and reported progress early in October, 1926, when the condition was found unchanged. She had noticed nothing unusual with regard to her vision in the interval. Mr. Lawford saw her, and confirmed the diagnosis, and on December 2 the right eye was trephined with a small basal iridectomy. The only noticeable thing that occurred during the operation was an undue amount

of oozing of blood while the sclero-cornea was being cut by the trephine. The case did perfectly well.

(7) This lady, the superintendent of a nursing home in London was under the care of Mr. Foster Moore. Her age was 56 years, and she first had trouble with her eyes in April, 1923, when she complained of seeing a black speck before the left eye. This symptom apparently disappeared, and in May, 1924, she had a recurrence of symptoms, and complained of having had several attacks of defective sight, and she also found that she had often run into people because she could not see them. Again these symptoms appear to have subsided.

Mr. Foster Moore saw her for the first time on October 27, 1925. Her visual acuity, with correction, was 6/6 in each eye, the tension of each eye was increased, the discs were cupped, and arterial pulsation was present. After consultation with Sir John Parsons, a trephine operation was performed on each eye; the first on November 7, on the left eye, and that on the right, on November 14. When first seen practically the whole of the upper part of the field of the left eye was absent, the defect reaching at one spot almost to the fixation point; the right field showed a defect typical of chronic glaucoma, reaching to within 10° of the fixation point.

She made an uninterrupted recovery, and when last seen on November 9, 1926, her visual acuity with +2.0D. sph., was 6/6 in each eye. The visual fields, at this date, showed considerable improvement; in the left eye she had recovered an area of considerable size in the upper temporal field, while in the right eye the defect, though still marked, nowhere came nearer than 20° to the fixation point.

(8) This lady, a missionary, home from China, aged 54 years, was under the care of Sir John Parsons. There was a six months' history; she had noticed that when reading, the lines above those at which she was actually looking, appeared crooked, and those below also. The vision had become dimmer of late. These symptoms were located in the left eye; she was not conscious of any disability in the right. The eyes externally looked healthy, the pupils were equal and active, the tension in the right eye was normal, while that of the left was very slightly above the normal.

The vision was as follows:

Right eye: 6/6, +0.5D. sph. 6/5.

Left eye: 6/9 part, +1.0D. sph. 6/6.

With +2.0D. sph. added, she read J.1. Each disc was deeply cupped; the left field of vision was contracted almost to the fixation point, the right showed the usual defect in the nasal area. She was admitted to the Royal London Ophthalmic Hospital, and on April 21, 1926, the left eye was trephined, and five days later the same operation was performed upon the right eye. She

was discharged on June 10, with good central vision, and fields contracted almost to the fixation point in an upper and inner direction. Mr. Foster Moore had an opportunity of examining this case, and found her exceedingly like his own, previously described, only not so far advanced.

(9) This gentleman, aged 53 years, was seen by me on February 12, 1926. He had joined up for war service and had had a remarkably fine war record, having won the D.S.O. When seen he was bordering on a nervous breakdown, due to the illness of his wife. There was a history of very bad vision, without pain, in the left eye of two years duration. His doctor had advised him to see an oculist, but he had omitted to do so, and an optician had informed him that in his opinion the left eye had been defective since birth. Questions after the examination elicited the fact that he had noticed halos since 1918, but as he had had no pain he had paid no attention to them. The right eye had recently showed signs of failure, also painless.

Externally the eyes looked healthy; the right pupil was active and the tension was normal; the left pupil was inactive to light and the tension was slightly raised. His vision was as follows:

Right eye: 6/6 part, not improved with glasses. J.1. with +1.5D. sph.

Left eye: bare perception of light.

The right disc showed distinct pallor, the vessels tilted towards the nasal side, there was a steep edge to the not very deep cup, upwards. The field showed a typical contraction, reaching to within less than 10° of the fixation point. The left disc showed a typical glaucoma cup and was very pale; there was arterial pulsation present, and there was a small retinal haemorrhage below the disc. He was ordered to use eserine and reported in a month's time that his sight was not so good. Mr. Lawford saw him in consultation next day, found the tension distinctly raised in the right eye and advised early operation. On March 18 the right eye was trephined with an iridectomy, the coloboma resulting therefrom being nearly complete. The sclerotic was rather vascular. On leaving the nursing home at the end of March the tension was soft and the vision was 6/12 −0.5D. sph. =6/6. When last seen on June 11, 1926, everything was satisfactory, the vision with −0.5D. sph. 6/6, the tension, soft, and the field showed a definite though slight improvement.

(10) This lady, aged 50 years, is a patient of Mr. Wardale of Newcastle-on-Tyne. She complained of no symptoms and consulted Mr. Wardale on account of the family history. She was a simple presbyope and the fields showed no abnormality.

(11) This lady is also under the care of Mr. Wardale. She is aged 48 years. She was seen by him in 1924, when spectacles

were necessary to enable her to do clerical work with comfort. Mr. Wardale writes to tell me that he saw her twice last year. She complained of occasional halos round a light; these were momentary and if she turned her head away and then looked at the light again, the halo had disappeared. Her central vision was found to be normal and she required a weak spherical glass for reading; the fields were found to be quite normal.

(12) This gentleman, aged 46 years, was under my care. He had been wounded in the left side of the neck during the war, and had a paralysis of the sympathetic nerve on that side. He had had no symptoms and came to be reassured in view of the family history.

Apart from signs due to the sympathetic palsy his eyes showed no abnormality on inspection. His vision was as follows:

Right eye: 6/6 part, +0.25D. sph., +0.25D. cyl. axis, 90°, 6/5 part.

Left eye: 6/5, +0.25D. sph. +0.25D. cyl. axis 90°, 6/5.

J.1. with +1.0D. sph. The fundus showed no abnormality, physiological cups were noted and the scleral ring appeared to be rather more marked in the right eye than in the left. The left field and blind spot were normal; that of the right showed a very slight defect up and in, reaching, on going over it again more carefully, to within 30° of the fixation point; it reminded one of the centripetal fan scotoma described by David Priestley Smith in the *Brit. Jl. of Ophthal.*, Vol IX, p. 233. The blind spot also showed a very slight general increase. He was ordered eserine, and in October he saw Mr. Lawford, who agreed with the diagnosis and advised operation at no very distant date. He came into St. George's Hospital and had a trephine operation performed on October 27. Everything went off satisfactorily, a small basal iridectomy being done. He left hospital after a fortnight and when last seen on December 17 the result was found to be satisfactory. It might be added that when seen on entering the hospital a definite increase in tension in the right eye was observed.

(13) The youngest son, aged 42 years, was under my care. He was in the Forests Department of the Indian Government and was home on leave. He came to be reassured in view of the family history and was found to be normal. He had had no symptoms and there were no signs of glaucoma, vision being 6/5 in each eye, with full fields and no enlargement of the blind spots.

It should be added that case No. 9 has two sons who are at school. His wife died in the summer of 1926.

In this family we have personal observations on six cases of chronic glaucoma, the eldest and the youngest sons are known to be free from the disease, as also two elder sisters. The

disease has been of a peculiarly insidious nature and has not, as far as appears, been rapidly progressive in any of the subjects. Cases Nos. 6 and 10 would certainly have been missed by the observer if he had not known the family history, and it might be added that case No. 9, when seen for the first time, entirely omitted to mention the fact that his sister had recently had each eye operated on for glaucoma and that his mother had suffered from the same disease.

I owe a debt of gratitude to Mr. Fisher, Sir John Parsons and Mr. Foster Moore who have collaborated with me in the preparation of this paper and to Mr. Wardale for kindly sending the notes of his cases of which previously I had no knowledge.

A FAMILY HISTORY OF CHOROIDAL SARCOMA

BY

R. C. DAVENPORT

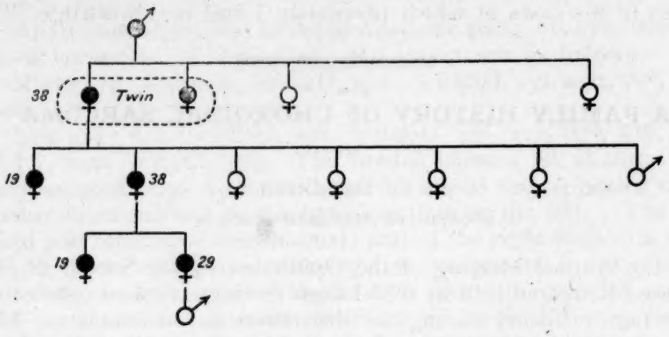
MOORFIELDS RESEARCH SCHOLAR

AT the Annual Meeting of the Ophthalmological Society of the United Kingdom held in 1926 I drew attention to the existence of this family history during the discussion on melanomata. The record is now so striking and so complete that I have thought it worth setting out in full. Two generations have been reported previously in Moorfields' records and more fully by Sir John Parsons in the *Trans. Ophthal. Soc. U.K.*, Vol. XXV, but I am able to add two females of the third generation whose records are at Moorfields.

The first case was treated in 1871, a woman, aged 38 years, whose left eye was excised for sarcoma. The case is reported by Collins, Lawford, and Marshall in the *Roy. Lond. Ophthal. Hosp. Reps.*, Vol. VII, p. 389; Vol. IX, p. 42; Vol. XIII, p. 117 (Case 2). She is known to have died seven months after operation from secondary deposits. All this is definite, and there is some less definite evidence showing that her father had an eye removed for some condition, not an injury, and that her twin sister had an eye removed at St. Bartholomew's Hospital, again for something other than injury. Another sister had a tumour removed from the breast at the age of 40 years. I can get no further details of these members of the family. Two daughters of this first case suffered from choroidal sarcoma, the younger having her left eye excised (in 1899) at the age of 19 years, which eye on section contained a typical sarcoma (Collins and Lawford, *Roy. Lond. Ophthal. Hosp. Reps.*, Vol. XIII, p. 117; Case 99). Five years later she died from secondary

deposits of the growth. An interesting feature in her case is the fact that when she was born her mother undoubtedly had the sarcoma present in her left eye. The elder daughter at the age of 38 years, in 1904 had her left eye removed, again the section showing a sarcoma present of an unusual type, fully reported by Sir John Parsons. She actually had secondary deposits present while in the hospital and died in 1905.

More recently two of her daughters each have had an eye removed at Moorfields for choroidal sarcoma. In each case it was the right eye. The elder had her eye removed at 29 years of age



● = Known cases of choroidal sarcoma

● = Suggestive history

in 1918, and, though she kept well for some long time, there was later a rapid dissemination of the growth in many organs and she died in 1922. Her younger sister had her eye removed in 1914 at the age of 19 years. On section it was found that there was an extra-ocular extension of the growth close to the optic nerve head measuring some 4-5 mm. in diameter. In spite of this there has been no recurrence of the growth locally. She has kept in good health and attended her sister in 1922. In 1923, a "lump" appeared beneath the skin of the right arm and this was excised at a London hospital as a ?lipoma. On section it was found to be a typical metastasis of melanotic sarcoma. No further trouble was noticed till 1925 when she was re-admitted there for ?adenitis in the axilla, but no tumour was found, for excision. Later, after discharge, this trouble recurred near the left breast, and another "lump" appeared on her back, and she is now under treatment at the Homoepathic Hospital for the "lumps" though apparently well in herself. The lumps are subcutaneous, sharply defined,

rather harder than lipomata but elastic, freely movable, painless, and have appeared stationary for over twelve months. She is not losing weight or showing any other signs of dissemination of melanomata.

The diagram of the family tree shows the sequence through the generations, but apart from that striking feature there are other points of interest, *viz.*, the ages; the average age for the five recorded cases being 28.6 years, and the fact that all have died from secondary deposits except one, who after ten years has now shown evidence of dissemination.

It is fortunate that the cases have all come to Moorfields and that, therefore, the clinical and pathological records can be assembled. As Moorfields Research Scholar I have been able to collect the records and in addition to see the earlier reports on the family and Sir John Parsons's note on the history. I am indebted to Sir Arnold Lawson and Mr. Hudson for permission to add the two later cases.

A FAMILY WITH BLUE SCLEROTICS

BY

MAJOR J. N. DUGGAN, D.O.(Oxon), F.C.P.S., L.M. & S.(Bom.),

AND

B. P. NANAVATI, D.O.M.S., M.R.C.S.(Eng.)

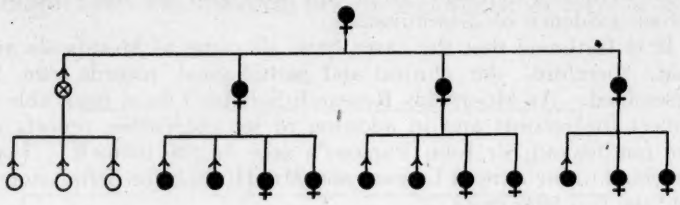
STRAY cases of blue sclerotics are often met with and occasionally found running in a family, but a case of blue sclerotic, described below, with a light blue discolouration of half of the face on the same side as the affected eye is of less frequent occurrence. We therefore include a description of this case along with the hereditary group.

The following are the notes on the various cases examined:

A Mahomedan boy, aged 11 years, was brought to the Sir C. J. Eye Hospital, Bombay, to have his vision tested, as he had poor sight. On noticing the blue colour of the sclerotics his father, who had accompanied him and whose eyes were normal, was questioned about their presence in the family. On his answering in the affirmative the affected persons were examined.

In the family these could be traced only on the side of the boy's mother. He had two sisters who were also affected. The mother of the three children had also blue sclerotics. Her brother who was slightly affected and both her sisters had the same colour about their eyes. The brother had three sons with normal eyes, but both the sisters had the abnormality in their families which

consisted of two boys and two girls in each case. On inquiring further it was found that the maternal grandmother of the children, who seemed to be the starting point, was also similarly affected. This brought out the interesting points that the abnormality was transmitted through the affected females to the descendants of the family and was dying out through the only male member who was slightly affected. There was no consanguinity of parents. The family tree is as follows:



Affected male = ♂ Normal male = ♂
Affected female = ♀ Slightly affected male = ⊗

The details of the clinical examination of some of the cases are as follow:

(1) A boy, aged 11 years, had both his sclerotics of deep blue colour. The greatest depth of colour was noticed in a circular zone one cm. wide round the limbus. The anterior perforating ciliary vessels were well marked out in the right eye with brownish pigmentation at the sites of perforation. He had myopia of moderate degree and visual acuity was 6/6 with sphero-cylindrical glasses. Iris was of deep brown colour. Fundi were normal.

(2) The colour of both the sclerotics of a girl, aged 10 years, was deep blue. The sclero-corneal junction stood out prominently as a white circle. Sites of the perforating ciliary vessels were marked out by deep brown pigmentation. On the surface of the right eye there were three small irregularly scattered patches of brown pigment. Vision and fundi were normal. Colour of iris was brown.

(3) This patient was a girl, aged 9 years. The colour of both the sclerotics was pale blue with scattered patches of deeper tint. Vision and fundi were normal. Colour of iris was brown.

(4) The mother of the above-mentioned children had a zone of deep blue sclerotics varying irregularly from 0.5 cm. to 1 cm. in width round the limbus and gradually fading into the light blue colour of the remaining sclera. Vision and fundi were normal. Iris was of dark brown colour.

The case of blue sclerotic with blue discolouration of the face mentioned in the beginning of the article was in no way connected with the hereditary group already described. It was noted in a Multani girl of a blonde type, aged 9 years, the sclerotic of whose right eye was of deep blue colour and who had an adherent leucoma of the same eye. The right half of the face had a faint blue discolouration. The discoloured area extended a little beyond the middle line, nearly up to it on the lower jaw and as far as the right ear. The right side of the nose and upper lid had escaped the discolouration. At the periphery the blue colour gradually faded away into the normal tint of the skin. The skin in the affected area was not thickened and did not show any dilated blood-vessels. The eye except for the adherent leucoma and blue sclerotic was of normal size and shape. No other member of the family was similarly affected though she herself was born with it. Before a photograph of the face could be arranged for she disappeared.

Remarks

(1) All cases of the hereditary type were bilateral.

(2) No evidence of fragilitas ossium, deafness, or congenital syphilis could be obtained.

(3) All cases were free from error of refraction except one that was myopic.

(4) All the affected members of the family were females with only one exception, that being a slightly affected male.

(5) The affection was transmitted through females to the descendants and was dying out through the only male member of the family.

A FAMILY WITH ANIRIDIA

BY

MAJOR J. N. DUGGAN, D.O.(Oxon), F.C.P.S., L.M. & S.(Bom.),

AND

B. P. NANAVATI, D.O.M.S., M.R.C.S.(Eng.)

IT is not often that one comes across cases of aniridia, much less so as a familial disease, and hence the following cases are considered worthy of record:

Family History.—All the affected children were the offspring of the third wife of their father, those by the first two being quite

normal. There was no history of any eye trouble on the father's side. The mother who died when she was aged 44 years, had nystagmus and poor sight. Their maternal aunt, who is still alive, was said to have gone blind a couple of years ago, but no evidence of nystagmus could be gathered from the lady who brought the children to the Sir C. J. Eye Hospital, Bombay. There was the history of a single miscarriage.

The family consisted of one girl and three boys. All of them had aniridia and varying degrees of nystagmus with myopia.

Case No. 1.—Miss H.K., aged 9 years, whose eye looked rather prominent, had bilateral hereditary horizontal nystagmus of moderate rapidity of movement. Cornea was normal. Only a narrow band of iris about 1 mm. wide, could be made out in each eye. Visual acuity of each eye was found to be 6/24, improved to 6/18 partly with -2D. sph. and -3D. cyl. axis horizontal. Ophthalmoscopically a very narrow rim of iris was seen, though in the left eye between five and seven o'clock it was wider than elsewhere. Lens of each eye was clear and its border round and regular. Efforts were made to see the fibres of the suspensory ligament and ciliary processes in the circumlental space which could be easily widened by making the patient look down, but neither could be made out with the ophthalmoscope. There were no vitreous opacities. Each fundus was normal and did not show any pigmentary disturbance anywhere.

Case No. 2.—Master P.K., aged 14 years, presented the appearance of aniridia in each eye, though the rim of iris, especially that of the left eye, was wider than that of case No. 1. Though no nystagmus was present, by making the patient look in the extreme horizontal positions, several nystagmoid jerks could be obtained in each position. Tension was normal. Visual acuity was 6/6 with -6D. sph. With the ophthalmoscope, a narrow rim of iris, which was wider in the left eye between five and seven o'clock than in the other part of the circumference, was seen. Each lens was clear and its border was regular and round. Fine vitreous opacities were noted. No suspensory ligament or ciliary processes could be made out. Both fundi were normal and there was no pigmentary disturbance.

Case No. 3.—Master A.K., aged 17 years, had bilateral horizontal hereditary type of nystagmus. Right eye, the cornea was of normal size. No rim of iris was seen in this case. The diffusely opaque lens with a regular border was subluxated upwards and inwards, but was not tremulous. A black crescentic area below the lower border of the subluxated lens attracted attention. Left eye, the cornea was faintly opaque and was larger than the right. The sclerotic was blue and the eye which as a whole looked

enlarged was congested and resembled a case of buphthalmos. The opaque lens could be seen moving about behind the cornea and seemed attached to the eyeball on the upper and inner side. There was xerosis of conjunctiva of both eyes. Tension of the left eye was much higher than that of the right.

His right eye previously had good vision, but for the last year and a half it had been failing. No reliable history of the vision or of any inflammation of the left eye could be obtained. On examination, the right eye was found to recognize hand movements and the left eye was totally blind. Formerly he was using -6D. sph. before the right eye.

With the ophthalmoscope, in the case of the right eye, a faint fundal glow could be obtained through the crescentic area below in which no rim of iris was seen. With the eye looking down, a white patch of fairly large size and irregular appearance with several white dots without any pigment scattered round it could be seen in the periphery of the fundus (probably choroidal atrophy). Projection was absent above. In the left eye there was no perception of light.

Case No. 4.—Master K.K., aged 11 years, had interstitial keratitis and aniridia in both eyes. He had a very rapid bilateral nystagmus of horizontal type. The vision of the right eye was fingers at four feet, not improved with glasses, and that of the left eye fingers at ten feet, with -11D. sph., 6/60. Ophthalmoscopically the left eye showed a narrow rim of iris. Fundus and media were normal. There was no pigmentary disturbance in any part of the fundus.

Wassermann was negative in all cases.

Teeth in all cases were found to be carious and misplaced but not notched. There were no signs of congenital syphilis.

Summary

(1) All the cases of aniridia seemed to have inherited the defect from their mother who had nystagmus, bad sight, and probably aniridia.

(2) All the cases had myopia and nystagmus except one in whom nystagmoid jerks could be obtained.

(3) In none could suspensory ligament, ciliary processes, or pigmentary disturbance in the fundus be seen.

(4) In one case interstitial keratitis was present. In another boy subluxated opaque lens in each eye and buphthalmos on one side were found. In the last case, a white patch probably of choroidal atrophy was noted.

KRUKENBERG'S SPINDLE

BY

ERNEST THOMSON, M.D. AND A. J. BALLANTYNE, M.D.

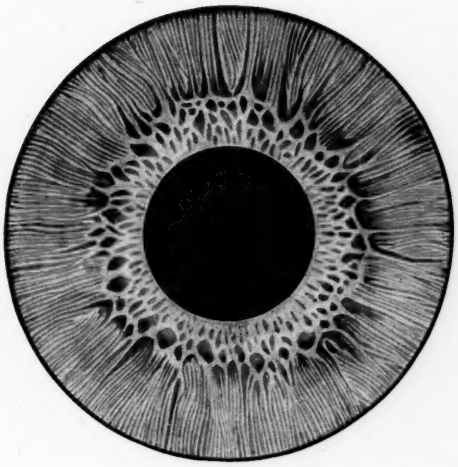
STIRLING GLASGOW

IN view of the renewed interest in this abnormality due to the publication in this journal of articles by Cardell and by R. R. James, we think it worth while to refer to a case of congenital bilateral pigmentation of the cornea which we published in the *Trans. Ophthal. Soc. U.K.*, 1903, and which was there stated to be a condition "apparently identical with that described by Krukenberg and by Stock." The drawing (by Ballantyne) was not published in the *Trans. Ophthal. Soc. U.K.*, and it is mainly for that very important reason that we again present the case. In describing it we cannot do better than quote from the *Transactions*.

The patient was a myopic girl, aged 22 years; the corrected visual acuteness was 6/9 and J1. with either eye.

"In the middle of each cornea there is a faint brown vertical line. On oblique examination, especially when assisted by magnification, it is seen that this line is composed of a great number of minute, chocolate-coloured dots. The largest and densest mass of dots is opposite the centre of the pupil. From this point downwards for a distance equal to one-fourth the corneal diameter the dots are fewer and spread out like the tail of a comet, the centre of the tail being denser than its edges. The pigment is interstitially placed, but its exact depth is difficult to determine." (At that date slit-lamp examination was not available, and our estimate of the position of the pigmentation was possibly faulty.) The history obtained from the mother was entirely negative as regards birth injury, or previous inflammation of the eyes such as might account for the condition. "No trace of iris adhesion; indicating a previous irido-keratitis, can be found. The iris is normal in every respect and there are no visible remains of a pupillary membrane. There are no vitreous opacities."

We considered that we were dealing with a congenital abnormality and gave our reasons (*vide Trans. Ophthal. Soc. U.K.*), but had no explanation to offer as to how it occurs. Cardell in his paper says, "Krukenberg and Stock regarded the condition as being on the posterior surface of the cornea and derived from the pigment of a persistent pupillary membrane, which, once attached to the posterior surface of the cornea, had left the indication of its attachment." He seems to imply that Krukenberg and Stock, like Vogt, consider the condition to be acquired, using the term "acquired" as indicating that it resulted from disease, rather than from a developmental anomaly, although pre-natal in origin. In



KRUKENBERG'S SPINDLE. ×6 diam. (A.J.B.)

the majority of the cases referred to by Cardell the lesion was attributed to a developmental defect. Our case, in which there was no evidence of inflammation, probably belongs to this group.

REFERENCES

1. **Cardell.**—*Brit. Jl. of Ophthal.*, p. 198, 1926.
2. **James, R. R.**—*Brit. Jl. of Ophthal.*, p. 148, 1927.
3. **Krukenberg.**—*Klin. Monatsbl. f. Augenheilk.*, pp. 254, 478, 1899.
4. **Stock.**—*Ibid.*, September, 1901.
5. **Thomson and Ballantyne.**—*Trans. Ophthal. Soc. U.K.*, p. 274, 1903.

NOTE.—The recent article by Koby " Pathogénie de la pigmentation fusiforme de la face postérieure de la cornée " may be consulted with advantage. *Rev. gén. d'Ophtal.*, February, 1927.

ANNOTATION

Conservation of Vision Classes

The American National Committee for the Prevention of Blindness has recently sent us several of their valuable reports, on some of which we have already commented. Three of these reports deal with the management and formation of special classes in ordinary schools for those children whose sight is not sufficiently defective to necessitate their treatment in special schools, but who are unable to profit sufficiently by ordinary tuition, or who are in danger of having their defect made worse by too much close work. These pamphlets are not addressed directly to ophthalmologists, although containing much information of use to them, but to the teachers and school medical officers. Of the three pamphlets under discussion the most valuable is one by Miss Estella Lawes, which gives an account of the course of instruction given at the University of Cincinnati in 1925.

These classes are a new development in school life, and as ideas are constantly altering, it is important to remember that the subject is open to changes and improvements as fresh experience accrues. The booklet is one that should recommend itself to all ophthalmologists, and to teachers and educational boards as well. These classes are established (*a*) to conserve sight in those children whose vision may deteriorate under ordinary school conditions; (*b*) to afford education for children whose sight is so defective that progress is impossible without the use of large type textbooks and material; the cost of which on a large scale is at the present time prohibitive. The children have been divided into two groups: (1) those with progressive eye defects, mainly myopes, (2) those with permanently low vision.

The following points have to be impressed on the teachers: (1) These children are not blind, their chief avenue of perception is visual and not tactile, as is the case in the true blind; (2 & 3) the nature of the handicap and the mentality of the child.

The pamphlet covers a great deal of ground, dealing in turn with the class-room, illumination, desks, equipment, paper and pencils, size of class (the ideal to be aimed at being that one teacher "would be able to take care of possibly four grades, with a maximum of 16-18 children") and standards of work. The importance of oral instruction and work is insisted on, especially in dealing with mathematics. Under writing, the teaching of a "running print" is advised, in order that the child may avoid having to learn four different symbols of any letter, *i.e.*, a capital and small letter, both in script and print. An important section follows on the place of the typewriter in education of this sort. Then follow studies in regular classes, such as languages, history, geography, spelling, maps, globes, cooking classes, physical training, eye hygiene, and the luncheon hour. The final paragraph of the booklet emphasizes the fact that the ideas set out in it are tentative.

The second pamphlet (Conserving the Sight of School Children), also contains a useful chapter on the formation of these classes, while the third (The Sight-saving Class Exchange) is an account by Mrs. Hathaway and Miss Peck of myope classes in England and Scotland. This last gives, we fear, too favourable a view of what has yet been accomplished.

Kerr pointed out in his book on School Vision (*Brit. Jl. of Ophthal.*, Vol. IX, p. 477), that there are about two thousand children in London requiring education in the present myope classes, but there are at least ten times that number who would benefit visually by a simpler sight-saving education than is afforded by any arrangements in the elementary school. All of us who have done work for the educational authorities in this country are only too familiar with the second class. Excellent though the work done by the myopia schools is, their scope can at best be only a limited one for several reasons: expense, parental objections, lack of accommodation, etc. In any case they do not attempt to deal with Kerr's large second class, the class that will probably most repay attention. The arrangements suggested by the American report do not require specially trained teachers and can be carried on in the same buildings as the ordinary classes. The report advises that all children with vision between 20/70 and 20/200, as well as all children with progressive eye troubles, should be considered potential candidates for sight-saving classes. The details of such classes might be left to the individual ophthalmic surgeon responsible. The report recommends: "The equipment advisable for conducting sight-saving classes includes hygienic movable seats, adjustable desks, tables and

chairs, books in 24-point type, outline maps, heavy pencils, unglazed buff-coloured paper, typewriter and stand, material for handwork, correct natural and artificial lighting conditions, correct colour of walls and ceilings and matt finish for all surfaces." We consider that the institution of classes of this description by the various educational authorities would be of very great value, and would assist in setting the work of the school refractionist on a more satisfactory basis.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

AT the recent Meeting of Delegates at Scheveningen the following Regulations and By-Laws were adopted:

Regulations

Article I.—The aim of the International Congress of Ophthalmology is to further the progress of Ophthalmology and to provide an opportunity of meeting and discussion for those interested in Ophthalmology.

Article II.—The general control of the Congress shall be in the hands of an International Council, elected at a general meeting of members of the Congress. It shall consist of a Chairman, a Vice-Chairman, a Treasurer, a Secretary and eight members and shall hold office until the end of the next succeeding Congress.

Article III.—The time and place of meetings of the Congress shall be considered by the Council and proposed to and decided by the general meeting of the Congress.

Article IV.—Membership of the Congress shall be limited (1) to the holders of diplomas in medicine, and (2) to those whom by reason of their scientific eminence in any of the allied sciences, or of their services to the cause of Ophthalmology, the Council may consider to be desirable members. The Council shall reserve the right of veto in the case of any applicant.

Article V.—The subscriptions for members and associates shall be fixed by the Council in collaboration with the National Committee.

Article VI.—Relatives and friends of members shall be eligible for election as associates on payment of a reduced subscription. They shall not take part in any of the scientific or business meetings of the Congress, but shall be entitled to be present at the social gatherings.

Article VII.—The local arrangements for each Congress shall be in the hands of a National Committee, selected by the Ophthalmologists of the country in which the Congress is to be held.

The National Committee shall work in collaboration with the International Council.

Article VIII.—The President of the Congress shall be chosen by agreement between the International Council and the National Committee.

Article IX.—Secretaries, appointed by the National Committee, shall be responsible for keeping the minutes of the scientific meetings, for collecting the papers when read, and recording the discussions. They shall act as secretaries of the Editorial Committee.

Article X.—An Editorial Committee, appointed by the International Council, shall decide which papers are to be published in full and which in abstract, edit the discussions, and supervise in general the publication of the proceedings of each Congress.

Article XI.—A printed copy of the Transactions of the Congress shall be sent to each member.

Article XII.—The National Committee shall be responsible for the expenses of the Congress held under its auspices. After the business of the Congress is completed and the proceedings are published, it shall transmit audited and certified accounts to the International Council for filing in the archives of the Council. Any balance of funds after payment of all expenses shall be paid into the Council funds.

By-Laws

I. Original Papers.

(1) Any member desiring to read a paper must notify the subject and submit an abstract of the proposed paper to the Secretary not less than three months before the date of the Congress, so that it may be printed before the meeting.

(2) Abstracts shall contain only a summary of the main conclusions of the paper and shall be translated into at least three of the principal European languages.

(3) No communication which has already been published shall be read at the Congress.

(4) Ten minutes shall be allowed to the author for presentation of his subject and five minutes to each speaker in the discussion. (Members wishing to take part in the discussion shall submit their names in writing to the Chairman.)

(5) Speakers taking part in discussion of papers shall be responsible for preparing a summary of their remarks and giving it to the Editorial Secretary before the end of the meeting.

(6) All papers presented to the Congress shall be left in the care of the Secretary for submission to the Editorial Committee and, if accepted for printing, shall not be published elsewhere than in the

Transactions of the Congress. If the Editorial Committee decide that the paper shall appear in abstract only, the writer may publish the full paper elsewhere.

(7) Reprints of papers appearing in the Transactions may be obtained by authors at their own expense from the printers, and must be ordered direct from the printers on receipt of the proofs.

II. Symposia.

(8) Subjects for symposia at each Congress shall be chosen by the Council who shall invite a limited number of speakers to introduce these discussions. The invited speakers shall each be allowed fifteen minutes. Each subsequent speaker in the symposia shall be allowed five minutes. By-law (5) shall also apply.

III. Demonstration of Cases and Specimens.

(9) A special sitting of the Congress shall be devoted to the demonstration of cases, specimens, instruments, etc.

(10) All cases and specimens must be accompanied by a card giving the particulars intended for publication.

OXFORD OPHTHALMOLOGICAL CONGRESS
XVII ANNUAL MEETING

THE Seventeenth Annual Meeting of the Oxford Ophthalmological Congress was held at Oxford on July 7, 8, and 9, under the Mastership of Mr. Philip H. Adams.

The proceedings took place in the Department of Human Anatomy through the kindness of Professor Arthur Thomson, whilst Keble College, where members met informally at dinner on July 6, was again available for hospitality.

After a brief formal opening at 10 a.m. by the Master, a discussion was held on "The results of the Operative Treatment of Glaucoma." The opener, Dr. WILLIAM H. WILMER, Ophthalmologist-in-Chief to the Johns Hopkins Hospital, Baltimore, began with a reference to the work of von Graefe, who, in 1857, wrote concerning glaucoma "the absence of general agreement constitutes an hereditary evil of therapeutics only to be cured by slow degrees."—"The only treatment from which relief can be expected is one that diminishes pressure."

That both these propositions are true to-day is attested by the fact that over sixty operative procedures, with many modifications are on record, all of which are devised solely for the purpose of reducing intra-ocular tension.

An analysis of a few of the kinds of operations and their results as reported by various authors is as follows:

Iridectomy: 482 cases: successful, 63.3 per cent. No late infection mentioned.

Anterior Sclerectomy: (Lagrange), 777 cases: successful, 72.8 per cent. Late infection (based on 389 eyes), 1.3 per cent. Lagrange's own cases, 104: successful, 85.5 per cent.

Sclero-Corneal Trephining: (Elliot), 3,381 cases, not including 911 eyes operated upon in Egypt, successful, 77.6 per cent. Late infection 1.2 per cent.

Punch (Holth). Holth publishes the record of 200 cases in Christiania from 1920 to 1923. The percentage of successes is not given, but he states that there were no late infections, and concludes: "The lowering of the ocular pressure is as good as in the other forms of sclerectomy and, so far, no case of late infection has been observed."

Hagen records 52 cases operated by Holth, Hj. Schiötz, and himself. Of these he excludes 12, confining his tables to 40 cases of glaucoma simplex. Successful, 82 per cent. Butler reports 29 cases. Successful, 79.3 per cent. Late infection 3.4 per cent. Later, he reports 39 cases, successful, 66.7 per cent.

Iris Incarceration: 517 cases: Iridotasis and iridencleisis. Successful, 87.4 per cent. Late infection, 0.3 per cent.

Wedge-Isolation and *Small Flap.* Sclerotomy (Herbert). The author has given up the former operation for the small flap sclerotomy. The reports are too few to analyze. Butler quotes 7 cases (small flap). Successes, 50 per cent., but this in no way indicates the excellent results obtained by the skilful originator himself.

Cyclodialysis (Heine), 1,027 cases. Successful, 66.7 per cent. No serious complications mentioned.

Whilst he, the opener, employed iridectomy, anterior sclerectomy and cyclodialysis the trephine was more frequently used by him. Two series of his own trephine cases were then presented, many of which had been under his personal observation for many years.

SERIES I.—Of these 72 eyes, 76.3 per cent. of the whole number have been under observation from 10 to 15 years; 19.4 per cent. from 5 to 10 years, and 4.1 per cent. from 3 to 5 years. Successes, 84.8 per cent. Failures, 15.2 per cent.

SERIES II.—35 cases (private and free), trephined within two years (none within four months), are reported for comparison Successful, 91.3 per cent. Partial failure, 5.7 per cent. Complete failure, 2.8 per cent. In these cases success meant a tension of 18 or under (Schiötz reading) with a maintenance or improvement of the vision and the fields of vision as compared with that before operation.

Causes of failure were previous venous thrombosis, tuberculous

deposits filling trephine openings, traumatic dislocation of lens ten years after a successful operation, rapid development of immature cataract after haemorrhage, and repeated retinal haemorrhages.

In nineteen of the second series of cases a Tenon's capsule implant was made use of, a valuable procedure where the conjunctival flap is too thin and as a prevention against infection.

A series of very interesting slides of the eyes of rabbits and dogs was shown, upon which anterior sclerectomy, sclero-corneal trephine, wedge isolation, small flap sclerotomy, and iridotasis had been performed.

After commenting on the various types of operation the opener said that every operator is more skilful in the performance of the operation he selects, but for the average the trephine is more safely and easily performed than the Lagrange or punch operation. It is also suitable for more kinds of glaucoma and in eyes with very high tension. In the Lagrange operation, if the pressure is high, there is more danger of expulsion of the lens and loss of vitreous at the time of the operation or later. Few operators have the skill of the originator of the excellent operation that bears his name. When the surgeon has much operative material, so that he can perfect his technique in both the Lagrange and Elliot, the plan of several of my colleagues is practical, that is, when the tension is below 35 (Schiötz) perform an anterior sclerectomy; when above, a trephine.

Professor FELIX LAGRANGE (Paris) who was accorded a very hearty reception, referred to the time when he performed his operation of sclerecto-iridectomy for the first time in England at the Oxford Meeting held in 1906 and commented on the cordial relationship ophthalmologically between France and England.

With regard to results he had never seen infection follow his operation.

Mr. A. L. WHITEHEAD (Leeds) said that after trying other procedures his rule now was to perform iridectomy in case of acute glaucoma and trephining for chronic glaucoma.

An analysis of 120 of his private cases, chosen because the records were more complete than in hospital cases, was as follows:

of 38 acute cases,

57.9 per cent. were successful,

26.3 per cent. partially so and 15.7 per cent. were failures;

of 14 subacute cases,

85.7 per cent. were successful, 14.3 per cent. partially so, and no complete failures;

of 48 chronic cases,

87.5 per cent. were successful, 4.1 per cent. partially so and 8.3 per cent. failures.

Lt.-Col. H. HERBERT (Brighton) considered that successful results were obtained with his flap operation especially if massage was regularly applied afterwards.

With regard to iris inclusion he thought there should be a distinction made between disasters at the time of operation and simple failures to relieve tension, and he felt that there ought to be no danger of sympathetic ophthalmitis provided that proper aseptic precautions were undertaken before the operation.

His belief was that the future lay with the small linear wounds either with or without iris inclusion.

Mr. MALCOLM HEPBURN (London) said that his operative experience had forced him to the conclusion that not all forms of glaucoma could be explained on the same pathological theory, but that different pathological conditions accounted for the various types of the disease. It followed from this that the results obtained by applying one type of operation for all forms of glaucoma would be variable and it was necessary for the surgeon in order to be successful to select the type of operation suitable for the case he had in hand.

Good results would depend upon this and upon operating early, bad results upon operating late, unsuitability of operation, mistakes in technique and secondary complications over which one had no control.

Lt.-Col. R. E. WRIGHT, I.M.S., agreed with Dr. Wilmer in his support of trephining as opposed to cyclodialysis and was interested in the method described by him for obtaining an extra cover of tissue over the trephine hole.

He was in favour also of a broad iridectomy in cases where he feared a "desquamation of uveal tissue" or "iritis" as it is termed.

Massage was of value in all cases although there was really no necessity for this after trephine operations.

Dr. C. F. BENTZEN (Copenhagen) reported the results of 72 cases of trephining in which he had separated the conjunctiva from the limbus instead of from above as in the usual Elliot method.

Of the 72 cases six were those of buphthalmos, four of acute glaucoma, 14 of absolute glaucoma and 48 of chronic glaucoma.

No case of late infection had occurred.

There were poor results in the buphthalmos cases and also in those of absolute glaucoma.

In the acute cases the results were moderately good and in the 48 cases of chronic glaucoma there were 77 per cent. of successes.

Professor SZYMANSKI (Wilno) described his modification of Elliot's operation in which he made use of a large trephine, cutting obliquely a semi-circle of sclera which was then excised with scissors.

Mr. HARRISON BUTLER (Birmingham) referred to the mental complications which happened in operations on elderly decrepit patients.

In iridencleisis operations he had found that no result was obtained, *i.e.*, neither fall of tension nor any untoward complication.

After trephining, a shallow anterior chamber might follow, which if persistent led to the formation of a nuclear cataract.

He had observed the fall of tension in the fellow eye after a successful operation on the other.

Mr. DAVENPORT (London) referred to the analysis of some 536 cases of glaucoma, 405 being primary, which had been trephined at Moorfields by various surgeons in the last five years. The results obtained were according to vision only.

These were reported fully in the BRITISH JOURNAL OF OPHTHALMOLOGY of 1926. In two cases only of the 536 did mental disturbance occur, and there were 14 cases of late infection of which, however, only eight were definitely in connection with the trephine hole.

Dr. HARALD GJESSING (Drammen) spoke of having had 81 per cent. of good results by Holth's method, the cases having been under observation from seven months to 11 years.

Mr. J. GRAY CLEGG (Manchester) considered that detachment of the choroid was a much more frequent result than usually thought, as much in his opinion as even 10 per cent.

Mr. B. GRAVES (London) referred to cases of persistent low tension after operation, in one of which shrinking of the eye had followed.

Mr. D. V. GIRI (Eastbourne) in speaking of the fluorescine test for filtration commented on the variations in tension which occurred during the day.

Lt.-Col. A. E. J. LISTER (Bristol) asked for an expression of opinion on the question of low tension after operation, which he regarded as a very alarming complication.

Dr. WILDER (Chicago) said that in America as all operations had been tried, but that no definite conclusions had been arrived at, this was good evidence that none was perfect.

The best thing would be for one surgeon to perform all the various procedures for comparison.

He personally was in favour of the iris inclusion operation, having only had one unsatisfactory case in 100 operated on by this method.

In reply, Dr. WILMER said that he had differentiated the cases with meticulous care but had omitted the reading of it out of sympathy for his audience.

Expulsion of the lens after the Lagrange operation was, according to the literature, a not uncommon complication, and one such case had been recorded after a trephine operation.

He had never experienced separation of the choroid in his own practice but had seen two cases in consultation, both of which promptly got well.

He had had cases of very low tension after both trephining and the Lagrange operation but had never had an eye in which there was delayed formation of the anterior chamber.

In the afternoon of Thursday, after Professor F. LAGRANGE had read a paper on "The best procedure to adopt for fistulization of the glaucomatous eye," demonstrations were held in the Scientific and Commercial Museums.

Lt.-Col. R. E. WRIGHT, I.M.S., demonstrated Green's apparatus for intracapsular extraction of cataract, and his own method of blocking the main trunk of the facial to prevent squeezing in cataract operations.

He also showed a series of extremely interesting histo-pathological slides of orbital neoplasms.

Dr. J. BURDON-COOPER (Bath) demonstrated a new astigmatic Test Chart.

Dr. THOMSON HENDERSON (Nottingham) demonstrated in an ox eye the membranous nature of the ligamentum zonulae lentis by injecting air through the ligament into the canal of Petit, thus showing that it is a closed space.

Dr. H. H. TYSON (New York) exhibited a series of skiagrams of the optic foramen showing the variations in size and shape from the normal, and demonstrated the effect which the narrowing of the canal had upon the optic nerve causing atrophy.

Dr. D. V. GIRI showed a photograph of a useful apparatus for red-free ophthalmoscopy and a lid-retractor which raised the lids free from the globe as well as retracting them.

Mr. F. A. ANDERSON (Shrewsbury) showed an excellent operating light obtained by a series of small lamps.

Mr. B. CRIDLAND (Wolverhampton) showed a drawing with specimen and slides of a case of cholesterin crystals in the anterior chamber.

The chief feature of the proceedings on the morning of Friday was the delivery of the Doyne Memorial Lecture by Professor K. K. K. LUNDSGAARD, of Copenhagen, the subject being "The Pneumococcus in connection with Ophthalmology."

The Lecturer showed a chart of the incidence of the pneumococcus on the normal conjunctiva as determined by various observers since 1909, the latest record being that obtained by himself in 1925, namely, 6 per cent.

The various types of the micro-organism were described and charted according to their virulence.

Other charts were shown giving the incidence according to age and seasonal variation.

A very complete survey of the *rôle* which the pneumococcus plays in ophthalmology was given, and Professor LUNDSGAARD, who, at the conclusion of the lecture, was presented with the Doyne Memorial Medal for the year, is to be heartily congratulated on his contribution to the knowledge of the subject.

Other papers delivered on the same morning were by Dr. CARSTEN EDMUND (Copenhagen), on "Colour perception in normal and night-blind persons in various illuminations," by Lt.-Col. R. E. WRIGHT who gave a résumé of notes of a series of fifty cataract extractions with Green's apparatus by himself and K. KOMAN NAYAR, and by Dr. HOLGER EHLERS on "Experimental researches on the corneal vessels."

The afternoon of Friday was spent at the Oxford Eye Hospital, where a series of interesting cases was shown by the Master, Mr. P. H. ADAMS and by Messrs. HARRISON BUTLER and HARRIES JONES.

After the cases had been examined, Lt.-Col. WRIGHT'S paper given in the morning was freely discussed by Lt.-Col. HENRY SMITH, C.I.E., I.M.S.,ret., and many others.

On the morning of Saturday papers were read in the Department of Human Anatomy, as follow :—

Dr. J. BURDON COOPER: The Permanent and Evanescent Canals of the Hyaloid Membrane with a suggestion as to their function in the drainage of the eye.

Dr. MARGARET DOBSON: Insufficiency of Accommodation as an Important Factor in the Causation of Incipient Cataract.

Dr. HARALD GJESSING: Further investigations into the results of iridencleisis operations by Holth's method, particularly as regards the method combined with meridional iridotomy.

Mr. D. V. GIRI: (*a*) An instructive case of high hyperphoria remedied by operation. (*b*) A case of extreme micrography with comments on its physiological optics.

On Thursday afternoon the members of the Congress and their friends were entertained at tea at Merton College with Mr. and Mrs. DE BEER kindly acting as host and hostess.

The Annual Dinner of the Congress which was well attended was held in Keble Hall on Thursday evening with Professor ARTHUR THOMSON as chief guest.

Members who attended from abroad, in addition to those already mentioned in the programme, included Dr. H. FRIEDENWALD (Baltimore), Dr. BERNARD SAMUELS (New York), Dr. BIRGER MALLING (Bergen), Dr. A. VERWEY (Durban), Dr. R. G. STOTT and Dr. A. S. ANDERSON (Australia) and Dr. PANDIT (India).

The total membership of the Congress is now over 400 and the attendance this year was well above the average.

COUNCIL OF BRITISH OPHTHALMOLOGISTS

IN the course of enquiries with regard to certificates for blind persons the following statement drawn up by the Lady Almoner at the Royal London Ophthalmic Hospital was brought to the notice of the Council. The Council considers that the information contained in this statement may be of considerable value to ophthalmic surgeons, and has therefore obtained permission to publish it from the Committee of Management of the Royal London Ophthalmic Hospital.

Until lately it has been the practice of the surgeons of the Hospital to certify patients who are blind by signing a certificate to this effect. Such certificates are required by the patients themselves, by voluntary associations, and by government departments, for many and various purposes. There is, however, a growing disinclination among some of the members of the surgical staff to sign such certificates on the grounds that services for State purposes should receive financial recognition.

If such a principle is accepted, in general, by ophthalmic surgeons, it becomes immediately necessary to separate those applications that are required for State purposes from those required by charitable agencies.

A rough classification is as follows :

For Charitable Purposes

Voluntary associations require blind certificates before they can (1) send blind patients to convalescent or permanent homes, (2) provide dentures, spectacles or artificial eyes, (3) arrange for home visitors, extra comforts or regular lessons in Braille, or (4) post books from the library for the blind.

For Semi-Charitable Purposes

Blind certificates are required before arrangements can be set on foot to train adult blind persons in the existing workshops. These workshops (Swiss Cottage, Tottenham Court Road, etc.) are maintained by charitable organizations—but receive Government grants in aid.

For State Purposes

(1) The L.C.C., in conjunction with the Ministry of Health require a blind certificate before allowing blind persons to derive benefit from their home teachers' scheme. (This is purely a London organization and does *not* affect the Provinces.)

(2) Those persons anxious to apply for the Blind Old Age Pension must produce a certificate of blindness.

The issue is, therefore, only clearly defined as far as the Blind Old Age Pension Act is concerned. The ophthalmic surgeon is here definitely assisting the Government to carry out social legislation. There is a misapprehension current that the Act provides the payment of a £3/3/0 fee to an ophthalmic surgeon on every case. That is not so. The Ministry of Health is empowered to pay an ophthalmic surgeon's fee *only* on an appeal case, and then *only* if the Regional Medical Officer cannot make a decision.

The Usual Procedure

The determination of a claim to a pension under the Blind Persons Act, 1920, rests, in the first instance, with the local Old Age Pensions Committee. No public funds are available to defray the cost of providing evidence of blindness for submission to the Committee. Every applicant is given a written statement to this effect, namely—

"A claimant to a pension under the Blind Persons Act, 1920, does not fulfil the statutory condition of blindness unless he or she is 'so blind as to be unable to perform any work for which eye-sight is essential.' Neither the Committee nor the Sub-Committee have any power to defray any expense to which the claimant or any other person may be put in connection with the claim, or any expense in connection with any certificate or other matter."

The Ministry of Health is not notified of any straightforward case—the Pensions Committee being responsible to the Treasury. There is not even a representative of the Ministry of Health on that Committee.

In the event of an appeal to the Minister of Health against a Pensions Committee's decision, it is the practice of the Ministry to refer the case to the Regional Medical Officer of the district, and, if he is not able to give a final decision, then the Ministry will pay a fee to an ophthalmic surgeon with a request for an examination of the case supplemented by a report.

It has been argued that all applicants for the Blind Pension should be examined by the Regional Medical Officers. The impossibility of this will be obvious when it is realised that for the "London District" (which extends from the Wash to Portsmouth and includes the Metropolis) there are only *ten* Regional Medical Officers in the whole area.

The part played by the Blind Societies in the administration of the scheme is merely incidental. Cases that come to the notice of the Blind Society are assisted in form filling and with advice. The Blind Societies are instrumental in reducing the number of appeal cases, and the aged blind who obtain their help receive their pensions with the least possible annoyance and delay. Without such backing many old persons entitled to the pension would never

succeed in obtaining it, simply because they are physically and mentally incapable of carrying through the necessary complicated regulations.

In conclusion, it seems clear that (1) the 1920 Act would have to be altered before ophthalmic surgeons could claim, with any hope of receiving, a fee from the Ministry, (2) if certificates of blindness are refused, as a preliminary to bringing the matter to the notice of the Ministry, very great hardship indeed will be involved as far as old persons are concerned.

ABSTRACTS

I.—GENERAL MEDICINE

(1) **Müller-Dehan, A.—Method and indications of tuberculin treatment. (Zur Methodik und Indikationslehre der Tuberkulinbehandlung.)** *Wien. Klin. Wochenschr.*, Bd. XXXIX, August 12, 1926.

(1) **Müller-Dehan** refers to the many conflicting views and methods advocated by various authorities on tuberculin treatment. From all these conflicting opinions, and from his own observations, he has worked out a method which he considers satisfactory. In this connection it is necessary to consider the various possibilities of tuberculin treatment more accurately than is generally done.

The basic principle in this treatment is "Tuberculous Allergy," the fact that non-tuberculous individuals are entirely unsusceptible to even the largest dose of tuberculin, while the tuberculous individual is affected by it in many different ways.

In the following remarks the term "tuberculin" refers to the *old tuberculin of Koch*—a concentrated filtrate of a glycerine-bouillon culture of the tubercle bacillus. All other tuberculins are to be considered as in some way weakened, detoxicated, or less toxic than the original form.

In the first place it is necessary to refer to the dangers which may result. The cause of these dangers lies in the very great individual differences in susceptibility to tuberculin, and therefore the possible occurrence of severe focal reactions in unsuitable cases. It is necessary to differentiate between local reaction at the point of injection or inunction—general reaction—of which temperature rises are the most marked symptoms, and focal reaction in the diseased tissue. A few cases are cited showing the dangers of beginning with large doses, and he therefore states that all diagnostic or therapeutic tuberculin injections must begin with

small doses in order to make sure that one is not dealing with an individual who is extremely susceptible. This gives an indication for tuberculin treatment, to render a very susceptible individual non-susceptible. The method is as follows:

A von Pirquet reaction is first done, and this gives some measure of the cutaneous susceptibility, which bears a slight relationship to the general susceptibility. If the von Pirquet reaction be very marked, the first injection is 1/10,000 mg.; if the reaction be slight, 1/1,000 mg.; if these injections cause no reaction, then at intervals of two days the dose is increased to 1/100 mg., and then to 1/10 mg. If no reaction occurs with this last dose, then the patient cannot be regarded as being specially susceptible. If it is desired to continue the course, then doses of 0.5 mg., 1 mg., and 5 mg. may be given. A patient who shows no reaction to 5 mg., and who at the same time shows no clinical signs of severe tubercle, may be considered to be free from active tuberculosis. If at any point in this series of injections a reaction occurs, the test must be interrupted until all signs of the reaction have disappeared. The limiting dose thus obtained serves as the commencing therapeutic dose, which according to the intensity of the manifestations may be repeated, diminished, or increased. The usual interval between doses is three to five days, for large doses—over 5 mg., seven to ten days—a final dose of 1 gramme may be attained by this method. Each dose is generally a 50 per cent. increase on the previous one, but this increase and the time interval is entirely dependent upon the severity and duration of the reaction. In the absence of any reaction, the dose may well be doubled. As a rule, however, doses will have to be repeated or diminished, especially so when a condition of persistent high temperature, or focal reaction occurs, or when after several repetitions of the same dose a limit is found beyond which it is impossible to go. In such circumstances, a reduction of 1/10 of this dose can be given, and from this point the dosage once again increased. If in spite of this the appropriate dose cannot be reached, then recourse must be had to one of the weaker forms of tuberculin, or specific therapy given up for a while in order later on to resume the old tuberculin.

The results of this method are in many cases very good, but the cases of increased susceptibility are not the severe cases of tuberculosis, but more frequently the milder, although very resistant cases, as for instance, phthisis with slight local signs but with a constant rise of temperature, and general symptoms. To this group of over-sensitivity belong many of the cases of tuberculosis of the eye. In fact, the following statement may be made when, in the course of a chronic illness, an increased susceptibility to tuberculin is found which may be suspected to play a part

in the course of the disease, *i.e.*, that this should be removed if no definite contraindication exists.

Many regard focal reaction as most important. This is an inflammation affecting the diseased tissue, and which may produce a tendency to improvement. Tuberculin would appear to act as a specific irritant—in the first place, it produces an effect on the whole organism, and locally, after the period of inflammation has passed it produces a tendency to cirrhosis and fibrosis.

There is no doubt that in tuberculous cases focal reactions may be obtained by non-specific substances, apart altogether from the reactions obtainable by exercise or sunlight, which is really a mobilization of autogenous tuberculin—as for instance protein, *e.g.*, milk, injections. But these reactions differ quantitatively if not qualitatively from those caused by specific substances. To take an extreme case, a healthy individual will show a demonstrable reaction to say 3 c.cm. of milk, and a tuberculous individual to say 1/10 c.cm. But a healthy individual will show no reaction to 1 grm. of tuberculin, while many tuberculous individuals will show a definite reaction to 1/100,000 grm.—therefore, the statement may be made, that *as regards focal reactions, and only as regards these, non-specific therapy may be used,* but the difficulty of dosage is very much greater.

It is fundamental that the question whether focal reactions are desirable or not must be set in every case, and each case must be judged on its merits. In general it may be stated that acute and active processes do not stand focal reactions well, while the more chronic conditions stand them better. As far as method is concerned, the one given above serves quite well, and it is best to start from some limiting dose, and watch the reaction by all available means. From the very nature of focal reactions, it is obvious that the time interval must be longer, sufficiently long to allow all signs of reaction to disappear, and frequently better results will be obtained by fewer but more widely-spread injections, than by following a definite plan. As regards the special preparation to be used, apart from non-specific substances, it is best to begin with old tuberculin, but frequently it is found that recourse must be had to one of the milder preparations. Where focal reactions are desired, a preparation must be used which is capable of producing such reactions; but where focal reactions are to be avoided, it is impossible to find a preparation which is entirely safe. Nevertheless, in properly selected cases, this method can be very useful.

The third therapeutic aim is the production of allergy. An attempt is made to increase the production of antibodies in anergic patients. There is a general method: if an antigen be repeatedly injected in doses below the reaction threshold, a hypersensitivity

is obtained, and this method can be employed with tuberculin using doses between 1/100,000 mg. and 1/1,000 mg. at intervals of five to seven days. The difficulty lies in the fact that with increasing susceptibility focal reactions generally occur—and it is in these cases of severe tuberculosis that focal reactions are specially dangerous. It is due to the desire to attain this result of successful allergy that constantly new forms of tuberculin and new methods of application are invented. In this method of using tuberculin we cannot estimate the effect on the sensitivity and therefore cannot avoid the occurrence of focal reactions. Some methods of estimating the effect of these small doses have been put forward as, for instance, Wright's opsonic index, but on the whole they have been unreliable, and at the same time very troublesome to carry out. In order to avoid focal reactions, the cutaneous or intracutaneous method of administration has been introduced, the idea being that the skin renders the tuberculin harmless, or detoxicates it, while its power of stimulating the formation of antibodies remains unimpaired. It is very doubtful if such be really the case. It is more likely that all the effects of tuberculin are weakened by this method of administration. The advantages are the greatly increased comfort of the patient and the absence of the injecting needle. The disadvantages are the very uncertain dosage, and the ignorance of the quantity absorbed and acting; therefore, this method is suitable only for cases in which the size of the dose and the intensity of the focal reaction are not important, as, for instance, in glandular tuberculosis; for the more difficult cases the subcutaneous method is the more exact. As regards method of application, for antitoxic and focal reactions, inunctions with a tuberculin ointment of increasing concentration such as the liniment of Petruschky or the Ateban of Neumann, and, for allergic effect, the giving of equal doses of a skin tuberculin such as Dermotubin or Ektebin. The Ponndorf method in which a tuberculin is brought into contact with a large area of scarified skin is a very gross one. The uncertainty of dosage is very great, and therefore this treatment is only advisable in very mild cases.

By the mouth, Koch's tuberculin is entirely without effect, but there are some tuberculins which are stated to be effective by this means; the difficulty of dosage is greater still and the whole method is still for the most part untried.

The Partigen-therapy of Deycke. Much of this rests upon a series of hypotheses and assumptions. It is maintained that in the tubercle bacillus and in its culture, there are present both the dangerous toxins, and the antigens. The toxins cause focal reactions and are to be removed, therefore the water soluble portion which is supposed to contain the toxins is abstracted, and the remainder divided into three groups—an albuminous, a neutral fat,

and an acid fat group. It is very doubtful if fatty and lipoid antibodies really exist. It is also doubtful whether this method really extracts these bodies in unchanged form. Practically, the effect is that of a weakened tuberculin, and these partial antigens do produce rises of temperature, and focal reactions. *Therefore, it may be said that allergic methods have not yet attained a final form, and that they may be carried out by very small doses of a weakened preparation given subcutaneously, or by the cutaneous method.*

Of other specific methods such as the sera of Maragliano, or of Marmorek, little need be said, and the same applies to the immunizing bodies of Spengler. These are all really protein-therapy methods, using very small doses.

Of interest, is the attempt made by Friedmann to influence tuberculous processes by the administration of non-pathogenic strains of the tubercle bacillus as, for instance, those obtained from tortoises. Good results have been reported in surgical tuberculosis, and in other mild forms, but in the severer cases, this method shows no advantage over the older method.

The experiments with prophylaxis, such as those attempted by Langer and Calmette have so far, not yielded definite results.

To sum up:

(1) (*a*) It is essential to differentiate between the different objects of tuberculin therapy, or to obtain a relative hypersensitivity to tuberculin (antitoxic therapy, therapy of tuberculo-toxic diseases); (*b*) to obtain focal reactions; or (*c*) to obtain allergic effects, or the formation of antibodies.

(2) The relationship between the desired objective and the method of procedure, *i.e.*, the choice of the preparation, the dosage, and the method of administration.

(3) The necessity to be clear, in each case, as regards the relative danger of focal reactions.

(4) The determination of the initial dose in each case by a gradual method beginning with the very smallest doses.

(5) If in the course of an illness, a hypersensitivity to tuberculin is discovered, and if it appear that this is of importance in the course of the disease, this hypersensitivity is to be removed provided that there be no definite contraindication to tuberculin.

(6) The treatment by means of focal reactions may be very useful in certain carefully selected cases and with correct doses. It can be carried out with specific and non-specific substances.

(7) The allergic method has not yet found its final form. It can be carried out by the subcutaneous administration of very small doses of a weakened preparation, or by the cutaneous method.

A. H. LEVY.

(2) **Mills, Lloyd, M. D. (Los Angeles).—Ocular disease occurring in the course of non-dysenteric amoebiasis.** *Jl. of Amer. Med. Assoc.*, October 9, 1926.

(2) No bacterial causation can be found in a definite proportion of cases of chronic, recurring, and intractable ocular inflammations, and conditions which arise in association with gastro-intestinal symptoms. Irido-cyclitis, episcleritis, choroiditis, and conjunctivitis are chief among these conditions. A large proportion of these chronic ocular conditions, unyielding to local treatment, are found associated with heavy intestinal infections of protozoa and flagellates of proved tissue-invasive capacity, and nearly always with an accompanying chronic colitis. The sole addition of effective anti-amoebic treatment to the local therapy has resulted in the relief, arrest, or cure of most of the inflammatory ocular conditions of this type so treated, and gives ground to the belief that chronic disease in any of the ocular structures may arise directly or indirectly from the known congestive and ulcerative changes which exist in these cases of intestinal parasitism. In **Mills's** opinion any case of chronic, recurrent, or intractable ocular inflammation or disease, in which the elimination of all possible foci of bacterial infection, and the correction of diet brings no cure, should be considered as a case of parasitic infection of the intestine and should be treated as such forthwith, unless really skilled examination of the stools is available.

A. F. MACCALLAN.

(3) **Tiscornia.—Ocular complications of leprosy. (Complicaciones oculares de la lepra.)** *Arch. de Oftal. de Buenos Aires.* March, 1927.

(3) **Tiscornia** has a comprehensive article on the ocular complications of leprosy. There are two varieties of leprosy, the anaesthetic and the nodular. In anaesthetic leprosy there may be anaesthesia of the cornea, with the sequences which are to be expected. In nodular leprosy lepromata may develop in the episcleral tissue at the limbus, or on the cornea. Leprous nodules may occur primarily in the ciliary body, and be manifest by irido-cyclitis and keratic precipitates.

A. F. MACCALLAN.

(4) **Bhaduri, B. N. (Calcutta).—Irido-cyclitis in leprosy.** *Ind. Med. Record*, September, 1926.

(4) **Bhaduri** abstracts the literature of ocular lesions in leprosy, and refers to a paper by D. J. Wood, published in this journal in 1925. He reports three cases of irido-cyclitis in his own practice. His first case, a Hindu male aged 34 years, showed signs of a

mild iritis, but did not at the time disclose the fact that he had had cutaneous lesions; the local condition quickly yielded to the ordinary treatment; later he had a relapse, with the appearance of skin lesions, for these he received anti-leprotic treatment and no further eye trouble was noticed; there were no nodules in the iris and the cornea was not involved. The second case denied the presence of leprosy though Bhaduri had previously been informed by his physician that he was under treatment for this condition. He showed signs of iritis with corneal haze, a nodule was observed in the iris; treatment led to some local improvement but a year later he returned in a much worse state. The third case occurred in a medical practitioner, aged 43 years, who had noticed cutaneous lesions five years previously; in this case the cornea was extensively involved, the pupil was irregular, the lens partially opaque, and the vision reduced to counting fingers.

The author comments on the unusual appearances shown in his first case, where the attack was very mild and no nodules or involvement of the cornea were seen; he considers it possible that the relatively benign course in this case may be due to toxins produced by the bacillus circulating in the blood and not to the presence of the bacillus itself; it might even be due to intestinal auto-intoxication occurring in a leprotic subject.

R.R.J.

(5) **Dudgeon, Leonard S. and Urquart, A. L. (Pathological Department, St. Thomas's Hospital, London).—Lymphorrhages in the muscles in exophthalmic goitre.** *Brain*, Vol. XLIX, pt. ii, p. 182, 1926.

(5) **Dudgeon** and **Urquart** have examined the extrinsic eye muscles, as well as the heart and some of the skeletal muscles in nine cases of exophthalmic goitre, one of whom also suffered from myasthenia gravis. Lymphorrhages were found in eight of the nine cases and were most marked in the ocular muscles. The lymphorrhages were both small and large, the latter causing wide separation of the muscle fibres. The cell contents of the deposits were chiefly lymphocytes with some plasma and endothelial cells. The lymphorrhages were often near a blood-vessel. An illustration and a bibliography accompany the paper.

E.E.H.

(6) **Bailey, Hamilton (Birmingham).—The value of Loewi's mydriatic test in the diagnosis of acute pancreatitis.** *The Practitioner*, August, 1926.

(6) In acute pancreatitis Loewi found that the instillation of adrenaline into the conjunctival sac caused dilatation of the pupil. This effect of adrenaline is not observable in the normal eye in the absence of acute pancreatic disease.

The technique given for the test is as follows: Examine the pupils; into one conjunctival sac instil four drops of 1:1,000 adrenaline solution; wait five minutes, then instil another four drops and wait half an hour. In acute pancreatitis one often gets a positive reaction, namely, dilatation of the pupil. This dilatation is not infrequently eccentric, and often conspicuously oval in form. A negative result implies nothing, but a positive result in an abdominal case is practically pathognomonic of the disease. The mechanism of the test is not easily explained. The inflamed pancreas by some means renders the whole of the sympathetic nervous system very sensitive. This sensitization of the sympathetic may be produced hormonically *via* the secretion of the islets of Langerhans, or mechanically by pressure of the swollen pancreas on the solar plexus. When this sensitization has been brought about, adrenaline in the conjunctival sac detonates the ocular sympathetic, causing a dilatation of the pupil. The explanation of the peculiar eccentricity of this mydriasis is even more obscure. It seems possible that the adrenaline will tend to gravitate in the lower part of the conjunctival sac, and thus the sympathetic fibres to this part of the iris may be more strongly stimulated than the remainder.

In the cases under **Bailey's** observation, which gave a Loewi's reaction, the dilated pupil did not react to light. There was one exception in which the dilatation of the pupil was very marked, but the pupil reacted immediately to a bright light, and took some three minutes to dilate again when the light was removed.

The author submits that Loewi's test is of very practical aid in acute abdominal cases.

A. F. MACCALLAN.

(7) **Guibert (La Roche-sur-Yon).—Ocular pemphigus, acute and chronic. (Pemphigus oculaire aigu et chronique.)** *La Clin. Ophtal.*, July, 1925.

(7) **Guibert** after discussing the rarity of ocular pemphigus, the statistical incidence of which varies from one in ten thousand to one in twenty-five thousand, and of which Hebra did not see a single example in 200 cases of pemphigus in general, shortly describes three chronic cases and then passes on to two acute cases one of which especially is of great interest.

In the first case there was only one bulla in one eye which was rapidly followed by corneal ulcer and hypopyon. There was fever for two days. Local treatment had no effect, but large and repeated frictions with collargol in the axillary and inguinal regions rapidly brought about a cure of the ocular infection. There remained a single symblepharon between the inferior tarsus and

the cornea, and there have been several recurrences on the hands, but otherwise the patient has remained well for fifteen years.

The second case is as follows:

Miss P., aged 51 years, was affected by sunstroke while sitting on a verandah for an hour. It was excessively hot and she left the verandah staggering, with large red blotches on the face and with painful eyes. She was seized with intense headache and could not sleep. Next day after drinking a bottle of lemonade she was unconscious for eight hours. On the third day the body was covered with blisters from head to foot and she could not open her eyes. Not until a fortnight later was she taken to hospital and was there found to have a temperature of 40° C., while the body was entirely covered with crusts, the general physical condition being very poor. No exact diagnosis was made, but since there was an infection of some kind, an injection of "propidon" was given and was speedily effective. (The reviewer does not know the nature of this drug which is not given in the latest edition of Martindale and Westcott.) From the next day there was a fall of temperature and immediate cessation of the formation of fresh blisters. Then the crusts began to fall off. At this time Guibert was asked to see the case. The patient was unable to open the eyes, a condition which hitherto had been supposed to be due to oedema of the eyelids. The eyelids were found to be glued to the globe except for a distance of about 3 mm. or 4 mm. at the most, a space sufficient to enable the patient to see, and sufficient also to prevent her realization of the gravity of the ocular affection. It was now a month since the commencement of the disease: would it be possible to free the eyelids? In spite of the protests of the profoundly objecting patient, Guibert, every day for a month and a half, passed a lacrymal probe up and down to what he took to be the limits of the *culs-de-sac*, and swept it round in a semicircle. At the end of the time the eyelids were free. It was the only chance, for no autoplasty would have succeeded in the infected state of the patient. At first the haemorrhage was considerable but it gradually diminished and was reduced to a few drops of blood at the end of the first fortnight. At each operation iodoform ointment was introduced between the lids. In spite of this very drastic and painful treatment two symblepharons on each side remained at the canthi where it had not been possible to be so thorough as elsewhere. A year later Dr. Marc Landolt operated on these symblepharons which have not since recurred at the end of three years. As regards internal treatment mercury was not well tolerated and "cacodylate" was employed. The author describes the condition of the hair, nails, teeth, mouth, and nose. For these details the original may be consulted.

ERNEST THOMSON.

BOOK NOTICES

An Introduction to Clinical Perimetry. By H. M. TRAQUAIR. 264 pp. 164 illustrations and a coloured plate. London: Henry Kimpton. Price 30s.

This authoritative volume modestly termed by its author an Introduction to Clinical Perimetry, is in reality almost an encyclopaedia of the subject. Compiled very largely from the author's own observations it forms a notable addition to British ophthalmic literature.

The Edinburgh School of Ophthalmology has for a long time been noted for its valuable contributions to this subject, as is exemplified not only by the work of the author but by the earlier work of Dr. A. H. H. Sinclair.

The book opens with an introductory chapter on the field of vision, which is likened to a hill surrounded by a sea of blindness, the contours of the hill being given by the different isopters of the field. Such a conception is of great importance to the perimetrist, since for clinical purposes we may regard a field as normal only "when the extent of its boundaries and its sensitiveness over its whole area attains the standard found in the average healthy individual."

The next chapter concerns instruments and gives details of the various types of perimeters and screens. With regard to the former, the author is in favour of a simple type in which there is no carrier or self recording apparatus, the test objects of various sizes and colours being mounted on long black holders. For screen work, a square of black velvet is recommended one to two metres in size, preferably mounted on a frame, with a fixation point of white paper or cardboard, attached by a black-headed pin. The edge of the screen is marked at every ten degrees by a stitch of coloured wool, using different colours to mark the different meridians; the remainder is ungraduated. The field is marked out with small black-headed pins, and the result transferred to a chart by means of a tangent scale. The rest of part one concerns methods of examination and the physiology of the visual field in relation to clinical perimetry.

The remainder of the book concerns applied perimetry and details the field changes found in lesions of the visual apparatus, from the choroid and retina, back to the suprachiasmal pathway. There are also chapters on the pathological field. The latter contains an excellent account of those anatomical features of the visual mechanism which concern the clinical perimetrist. The course of the nerve fibres in the retina for example, is carefully traced, the presence of the macular raphé being explained by Roemer's

hypothesis, *viz.*, that it may be regarded as a fold in the temporal margin of the retina, drawn horizontally into the fovea, the edges of the fold falling into apposition so that the macula may be regarded as really situated on the temporal edge of the retina.

The reader of this book cannot help being struck again and again by the important part perimetry may play in all branches of ophthalmic medicine. There are many conditions, *e.g.*, glaucoma, in which perimetry is almost universally practised, but there are others in which it is hardly used at all and yet may be of great service. In myopia, for example, when choroidal degeneration is seen commencing on the outer side of the disc, examination with the screen will show whether this is progressive or stationary. In the former case, the central edge of the scotoma will be sloping or indefinite, whereas in the latter it will be sharply defined. Again, central vision in myopia may be quite good, and yet small white or coloured objects may be seen by the patient to "go in and out," indicating perifoveal disturbance, which may later extend to the fovea. In acute exudative choroiditis, field examination may be of service, in showing whether the inflammation has penetrated as far as the nerve fibres, since in the latter event, a nerve fibre bundle defect will be added to the scotoma produced by the destruction of rods and cones. In the diagnosis of early glaucoma, the importance of utilizing small objects may be shown by the following quotation: "Sometimes the isopter for 1/2,000 or 2/2,000 is contracted, mainly, or only, above, and normally situated below encircling the lower, but not the upper, part of the blind spot. The depression is not constant, but may be the only perimetric sign in eyes, known definitely, to be glaucomatous." In regard to the pathology of this disease, it is noted that the evidence of the scotomata suggests that it is not the nerve fibres, but their blood vessels which are compressed, in the first instance at any rate. In the chapter on the optic nerve, the author would seem to have introduced a new word "plerocephalic" oedema (from πλήρης, full to overflowing). His reason for doing so is that oedema of the optic nerve may occur from causes other than increased intracranial pressure, and that it is therefore convenient to have a word meaning oedema from this cause only.

The author's work on field changes in pituitary disease is well known to readers of this journal and his chapter on the chiasma is therefore particularly interesting. The fields are divided into two types, the scotomatous and non-scotomatous. The former is indicative of activity of the lesion, the latter of its being slowly growing or stationary. A full description is given of the progress of the condition.

After the chiasma, the suprachiasmal pathway is dealt with. In lesions of the optic tracts, the author notes that the hemianopia in

its typical form is incongruous, relative, incomplete and progressive and only in its final stage, complete and absolute, while lesions above the external geniculate body, produce congruous and simultaneous defects. The final chapter on functional changes in the field of vision is followed by an appendix giving statistical details of the isopters for white and colours in the normal field, the size of the normal blind spot and various other items of useful information.

In conclusion this is a book which should find its way into every ophthalmic surgeon's library, and may we hope that once there it will act as a stimulus to him to make the fullest use of the perimeter and screen in the examination of his patients.

La Tuberculose de l'Oeil et de ses Annexes. Par E. ROLLET et A. COLRAT, Lyon. Paris: Gaston Doin et Cie. 1927. Prix 60 francs.

This is the tenth volume of a series of short treatises on tuberculosis of various organs of man: two more of the same series are in the press.

In the eight chapters comprised in their work the authors deal seriatim with tubercle of the cornea, sclera, uveal tract, retina and vitreous, optic nerve, lids and conjunctiva, lacrymal gland and lacrymal passages. In the chapter concerning the uveal tract (which extends to approximately one third of the book) will be found a valuable section on the technique of tuberculin and radio-therapy. The latter of these methods of treatment is especially commended by the authors in cases of corneal, sclero-corneal, and iridic tubercle. They consider it the most effective and the least dangerous of the conservative methods.

While the contents of the volume are mainly clinical, each chapter contains a brief section on pathological anatomy, and in those dealing with the cornea, conjunctiva, and uveal tract, one on pathogenesis and experimental production of tuberculosis in these structures.

This work, intended mainly for ophthalmologists, contains much that will interest physicians, especially those in charge of tuberculosis clinics. The book is illustrated by 24 figures in the text and four coloured plates. It contains a useful though incomplete bibliography, an index and table of contents.

Chirurgie de l'Oeil et de ses Annexes. Par F. TERRIEN, Professeur de Clinique ophtalmologique à la Faculté de Médecine de Paris. Troisième Edition augmentée et entièrement refondue. Paris: Masson et Cie. 1927. Prix 100 francs.

In arrangement the 3rd edition of Terrien's Surgery of the Eye follows very closely the 2nd edition; the latter was reviewed at some length in this Journal. (See *Brit. Jl. of Ophthal.*, Vol. 5, p. 235.)

The new edition is larger by 26 pages and contains 69 more illustrations, while some of the figures in the former volume have been replaced.

The additions to the letterpress are chiefly in the chapter on cataract and in that on the orbit. The former contains a new section concerning intracapsular extraction of cataract by Smith's and Barraquer's methods, with illustrations depicting the steps of the operations. The author does not express any great enthusiasm for either method and says of intracapsular extraction generally, "C'est sans doute l'opération de demain, mais ce n'est pas celle à enseigner aux élèves."

The latter includes a new section on sphenoidal sinusitis and its treatment by Segura's method.

Minor additions and alterations are noticeable in other chapters, enhancing the value of the book and bringing it quite up to date.

In respect of printing both of letterpress and illustrations this edition maintains the reputation of its forerunner. The proof reader has been exceptionally vigilant.

CORRESPONDENCE

SYMPATHETIC OPHTHALMITIS

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—I have read in the annual report of the Ophthalmological Society, an account of the discussion on sympathetic ophthalmitis. It is a matter of regret to me that for geographical reasons I was unable to be present. In the circumstances may I venture, however, to make some comments on that discussion.

I have, in the course of 35 years or thereabouts, seen over 60 cases of sympathetic ophthalmitis. I agree with most of the speakers that there has been a great decrease latterly, and in the last 10 years, I have seen only one case. During the Great War, in the Egyptian theatre of operations not a single case was brought under my notice.

Various theories are offered for this decline in the frequency of infection, most of which appear to me to be unsound. Early excision of the injured eye was practised even more frequently 30 years ago than to-day. At present some of the younger ophthalmologists scarcely regard sympathetic ophthalmitis as a possibility and venture risks which were not usually taken 30 years

ago. Many writers ascribe the diminution to better asepsis in the treatment of wounds of the eye.

From my own experience, I do not think that the asepsis to-day is any more efficient than it was 30 years ago. Both then and now it was and is thoroughly applied in the hands of most practitioners.

What then, is the cause of the decline, about the existence of which everyone is practically agreed?

Somewhere about 1906 when the Wassermann reaction was making its appearance I began to be suspicious that certain cases were not sympathetic ophthalmitis, but were syphilitic. When in 1910 the Government of Victoria instituted the National Investigation into the prevalence of syphilis, by the wholesale use of the Wassermann method, it became obvious that some of the cases of supposed sympathetic ophthalmitis were really cases of syphilitic kerato-iritis.

Certain wounds of the eyelids and cases of operation such as iridectomy did badly. The wounds became infiltrated and the eyes and tissues irritable, and many of these cases were found to give a positive Wassermann reaction. I submitted one case of injury to one eye with apparent sympathetic ophthalmitis in the other, to a number of colleagues, most of whom thought it was a genuine case, whereas the condition proved to be syphilitic and gave way to treatment.

Mr. Browning, in the discussion referred to gives a blood picture for syphilis, not unlike that of sympathetic ophthalmitis, and the question not unnaturally arises, what is the relation between the two diseases, particularly as the use of arsenical preparations seems favourably to influence the course of sympathetic ophthalmitis.

At the same time I can hardly recollect a case of syphilitic iritis which has produced the intensely hard and rigid condition of the iris which is met with in cases of genuine sympathetic ophthalmitis, and consequently I still regard sympathetic ophthalmitis as a clinical entity.

Those who wish to appreciate the change which has taken place should read the report of the late Marcus Gunn published in the Royal London Ophthalmic Hospital Reports, 1887, pages 78 and 273. I doubt whether such a report could be prepared anywhere to-day because of the lack of material.

Perhaps I may be permitted to summarise the matter, as it presents itself to me, by setting out the problem in categorical form.

1. There is general agreement that sympathetic ophthalmitis is much less frequent than formerly.

2. Neither early excision of the injured eye nor better asepsis can account for the diminution.

3. Some of the cases supposed to be sympathetic ophthalmitis were syphilitic.

4. The treatment of syphilis is far more efficient than formerly and syphilis is the only one of the venereal diseases which appears to be declining both in prevalence and infectivity.

5. The blood picture in syphilis and sympathetic ophthalmitis is similar.

6. Is it possible that a syphilitic background makes true sympathetic ophthalmitis more possible?

It should be noted with interest that in 1887, Marcus Gunn strongly urged the use of mercury in sympathetic ophthalmitis.

Yours truly,

JAMES W. BARRETT.

MELBOURNE,
June 16, 1927.

KERATITIS DUE TO MUMPS

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—In the BRITISH JOURNAL OF OPHTHALMOLOGY, Detroit, of Lille, quotes a case of the above. Some years ago I saw a similar case, and there was no doubt as to the cause, the patient having had a typical attack of mumps which he caught from his sister. When I first saw him his vision was reduced to hand movements at two feet, and the cornea was completely clouded all over, no fundus reflex could be obtained, and there was considerable circum-corneal injection. The appearance was that of an interstitial keratitis with grey streaks radiating all over the cornea. The iris was not involved. The treatment was simply atropine drops, 1 per cent., and in the course of about a week the whole cornea cleared, and vision became normal.

These cases must be rare, as I remember writing to Sir Arnold Lawson at the time to ask him if he had ever seen a similar case, and my recollection is that he said he had not, and very kindly looked up the literature for me, with the result that he could only find I think five cases quoted and those by a French oculist, whose name I have forgotten. It is possible that he may remember the facts. In view of the case mentioned, I thought mine might be of interest.

H. GOODWYN.

NEWBURY,
BERKS.

CATARACT EXTRACTION

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—I wish to call attention to some points arising out of the very interesting article by Major M. M. Cruickshank, I.M.S., on the complications following 4,000 cases of cataract extraction, mainly in the capsule, at Dr. H. T. Holland's clinic in Shikarpur, Sind, India, during the 1923, 1924 and 1926 seasons. In the section dealing with vitreous loss he asks "Is vitreous loss more liable to occur in the simple or in the combined operation?" and proceeds to give the following figures relating to this question :—

	Cases	Vitreous loss in	Per cent.
Complete iridectomy	650	85	13.08
Marginal iridectomy	144	0	—
Peripheral iridectomy	216	17	7.87
No iridectomy	240	19	7.91

He goes on to state "The marginal iridectomy was devised by Lt.-Col. Henry Smith and the majority of these cases were done by his son, Dr. J. R. Smith. . . . . It is noteworthy that in no single instance where it was performed did vitreous escape." The reader is thus led to believe, unintentionally I am sure, that the absence of vitreous prolapse in these cases was in some way related to the type of iridectomy performed, whereas in fact it had nothing at all to do with it. It was due to the fact that I was using the technique for expressing the lens, described by Col. Smith in the *Arch. of Ophthal.*, No. 3, 1926. In this, by external pressure alone, the lens is made to "tumble," *i.e.*, turn a somersault and come out lower edge foremost, the suspensory ligament remaining attached in the upper sector throughout the process until the lens is lying on the front of the cornea ready to be separated from its final attachments by passing the elbow of the squint hook under it along the wound.

In the section dealing with burst capsules Major Cruickshank quotes the same cases :—

	Cases	Burst capsules in	Per cent.
Complete iridectomy	650	41	6.30
Marginal iridectomy	144	0	—
Peripheral iridectomy	216	12	5.55
No iridectomy	240	9	3.75

Here again he discusses the relation of this complication to the type of iridectomy performed, whereas he omits to mention a factor which is in my opinion of infinitely greater importance—the size of incision used. In the cases done by myself, I paid particular

attention to making the incision of adequate size, *i.e.*, half the corneal circumference or a trace over rather than a trace under, and the influence of this can be seen in the fact that the 144 cases performed with a marginal iridectomy again escaped accident under this heading.

It would be interesting to learn how many operators by the capsulotomy method could report a series of 144 unselected cases dealt with *without accident on the operating table*, a matter in no way due to personal skill on the part of the operator—very far from it—but solely to the fact that with recent developments the technique of intracapsular extraction of cataract has been reduced to a simplicity which would hardly be credited by those who have not seen it with their own eyes.

Yours faithfully,

J. RUSSELL SMITH.

SIDCUP, KENT,
July 14, 1927.

NOTES

International Ophthalmological Council

THE Treasurer of the International Ophthalmological Council will be glad if any individuals who wish to subscribe to a fund for meeting the expenses of the Council will communicate with him, so that their names may be included in the first list of subscribers which will be published in a forthcoming number of the journal. Subscriptions on the part of individuals to this fund are entirely voluntary, and do not confer membership of the Congress. Institutes or societies subscribing £2 or more will receive a copy of the *Transactions* of the Amsterdam Congress of September, 1929.

Subscriptions should be sent to Mr. Leslie Paton, Treasurer of the International Ophthalmological Council, 29, Harley Street, W.1.

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North of England Ophthalmological Society

THE following are the officers of the North of England Ophthalmological Society for the Session 1927-28:—*President*: Mr. Thomson Henderson, Nottingham; *Vice-President:* Mr. D. Matheson Mackay, Hull; *Treasurer:* Mr. W. H. Kiep, Bradford; *Members of Council:* Mr. T. Louis de Courcy, Liverpool, Mr. W. Stirling, Manchester. The Secretary was re-elected (Mr. Percival J. Hay).